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To know Pittsburgh, you have to see it for yourself. Come and see a city that has had a remarkable environmental renaissance, a top-10 city for certified green building space, a city ripe with natural and cultural amenities.

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- In a statement to the New York Times, Prince Phillip said that Pittsburgh is the only city he has ever seen that has an entrance. The first glimpse of the Golden Triangle upon exiting the Fort Pitt Tunnel, on the way in from the airport, is absolutely incredible!
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Greetings! I hope each of you had a joyous holiday season with your family and friends. The big tree has come down but the rest of the decorations are staying up for a while. It is a season, not a day, and I like the decorations, and love the feeling of sharing, and giving.

Events for your consideration:

• The national AMT meeting is the week of July 8th in Pittsburgh, PA.

• Two openings will be available on the AMTIE Board. The terms of Marty Hinkel and Kay Fergason will expire. Marty is eligible for re-election, but Kay has reached her term limits and will not be running again. Please consider running for a position on the AMTIE Board. The AMTIE Board is a wonderful opportunity to solicit donations and give scholarships to members and students. A nomination form can be downloaded from the AMT website. The form must be completed and received by the AMT home office by April 1, 2013, in order to be printed in the AMT publication AMT Events. The deadline for declaring candidacy is July 8th at 10 am.

• Please consider donating to the Chester B. Dziekonski Memorial Fund when you pay your dues. Money donated is tax deductible. Monies contributed are for two continuing education/continued competency grants of $500. Grants are awarded at the national convention.

• Did you have fun during the AMTIE fund raiser last year in San Antonio? Another fun surprise is planned for you in Pittsburgh. Taffy Durfee has agreed to head up a silent auction. Georgia AMT member Peggy Oiler has suggested that members donate their custom crafts for this event. Many members seem to be excited about this fund-raiser and willing to show off their talents. I already know something I want to bid on. If you would like to participate and donate to the auction, please send Taffy a message at ndurf@st-joseph.org. We will again have the 50/50 raffle.

• The deadline for applications for the scholarships is April 1, 2013. If you are continuing your educational quest in one of the allied health careers that are certified by AMT or you know a high school student who plans to attend an AMT certificate program, go to the AMT website and download an application. There are separate applications for AMT members and students. The scholarships include one AMT member scholarship of $2,500, three AMT member scholarships of $1,500, and five student scholarships of $500.

As an AMT member, you have the opportunity to participate at the state society level and on the national level. Your efforts are appreciated!

You are the “Pride of the Profession.”

Best regards,

Linda
April 1 is the filing deadline for applications and supporting documents in the AMTIE 2013 undergraduate/graduate scholarship program and for grants to high school graduates pursuing medical technology, medical assisting, dental assisting, or phlebotomy studies.

Up to three $1,500 AMT Member Scholarships may be awarded annually. Applicants must be members in good standing with AMT and enrolled in a college or university accredited by a regional accrediting commission. The course of study should be concerned with the disciplines certified by AMT. Scholarship recipients will be selected by the AMT Institute of Excellence and scholarship committee based on financial need, career goals, and previous scholastic record.

Up to five $500 Student Scholarships are awarded annually and available to high school graduates interested in pursuing medical technology, medical assisting, dental assisting, or phlebotomy studies. Applicants must be enrolled or planning to enroll in a school accredited by an accrediting agency recognized by the U.S. Dept. of Education, or enrolled or planning to enroll in college, university, or junior college medical technology, medical assisting, dental assisting, or phlebotomy studies. Scholarship recipients will be selected by AMTIE Board of Trustees based primarily on need; taken into consideration are individual goals and motivation, school grades, participation in extracurricular activities, work experience, and personal references.

All scholarships will be awarded during the 2013 AMT National Convention in Pittsburgh, PA, July 8–12.

For information and to receive an application, visit www.americanmedtech.org

Application deadline is April 1, 2013.
It’s often said war comes with a price. In this case, one of the costs of war was the acquisition and ultimately the introduction of what some fear could cause widespread infection and death.

It’s multidrug resistant *Acinetobacter baumannii* and it was introduced into North America by troops returning from the Middle East in Canadian soldiers injured and requiring mechanical ventilation as a part of the medical treatment. The ventilators were populated with the organism that was passed along to the patients. In addition, soldiers acquired skin infections as a result of chafing with secondary infections. Already, tens of thousands of hospital patients have died from a result of infection.

Jason Tetro, microbiologist and Coordinator for Emerging Pathogens Research Centre and Centre for Research on Environmental Microbiology in Ottawa, Canada, said, “*A. baumannii* is ubiquitous in soil and surface water and can be carried on the skin by normal healthy individuals without any clinical consequences.”

“However, the bacterium is opportunistic and can infect individuals who have compromised immune systems. The rise in such individuals is primarily the reason for the increase in the case rate of these infections. As with any bacterium, an increased prevalence eventually leads to increased antibiotic resistance,” he explained.

Amesh Adalja, MD, FACP, clinical assistant professor at the University of Pittsburgh (PA) Medical Center said, “*A. baumannii* is on the rise for several reasons, the most important being the proportion of patients in hospitals who transition from hospital to nursing home and long-term care facility and back several times. He said these patients serve almost as vectors for the bacterium and introduce it into hospital settings which can then foster nosocomial spread through lax infection control practices.”

**The New MRSA?**

The similarities for the potential of widespread infection sound strikingly and alarmingly familiar to Methicillin-resistant *Staphylococcus aureus* (MRSA). According to the Centers for Disease Control and Prevention, MRSA is a type of staph bacteria resistant to treatment with a certain group of antibiotics called beta-lactams, including methicillin, oxacillin, penicillin and amoxicillin.

“The most ominous issue with *A. baumannii* is some strains are multi-drug resistant. We have no new drugs to combat it and, in some cases, there is nothing a physician can do to treat this infection,” explained Dr. Adalja.

From a purely infection control perspective, Tetro said healthcare practitioners may very well be staring down the barrel of the next strong, drug-resistant outbreak. “Hospitals are unfortunately the new breeding ground for infections and as MRSA started in health care facilities, so can be said for *A. baumannii*,” he warns. “Also, as MRSA became increasingly community-acquired, the same is occurring with *A. baumannii* albeit at a slower rate.” He said the problem for both these bacteria lies in the number of immunocompromised individuals who cannot co-exist with the opportunistic organisms and prevent infection.

Adalja is concerned, but not panicked. “I think in specific patient segments it could be akin to MRSA. However, I do not think this organism possesses the attributes necessary to cause a widespread epidemic in otherwise healthy individuals (unlike MRSA),” he noted. “*A. baumannii* infections principally occur in frail, elderly, chronically hospitalized individuals reflecting the predilection of this bacterium for a compromised host,” he said.

There are exceptions. In fact, some of those include military personnel with wounds. “But for the most part, *A. baumannii* would have trouble sustaining an epidemic in the general population. In the hospital — especially ICUs — it is considered on par with MRSA,” Dr. Adalja noted.

**Strict Infection Control**

Tetro said that, for the most part, all healthcare practitioners should be aware of the bacterium as a potential cause of illness.

“[Healthcare practitioners] especially need to understand the clinical symptomology of this infection,
which can range from UTIs to wound infection to bloodstream infections to meningitis,” he said. In the event they notice any possible change in a patient’s condition relating to these clinical impacts, they should be ready to explore the potential etiology of this bacterium.

Moreover, should *A. baumannii* be identified, Tetro said an immediate identification of any antibiotic resistance is necessary “such that proper stewardship can be performed.” Finally, any evidence of infection should be reported to the local health authorities for tracking.

Sheryl A. Whitlock, MA, MT(ASCP)BB, laboratory coordinator at the University of Delaware Student Health Services in Newark along with Kevin E. Whitlock, MT(ASCP)CM, a medical student at the Philadelphia (PA) College of Osteopathic Medicine, stress the increase in antimicrobial resistance may be related to overuse of antibiotics.

“Multidrug resistant organisms have developed and present a new public health hazard related to, but not exclusively caused by, extensive use of antibiotics. The unnecessary use of antibiotics has been addressed in the medical community extensively, but in some cases might be shutting the barn door after the horse is out,” explained Sheryl A. Whitlock.

“In addition to overuse of antibiotic agents, some patients who always want antibiotics do not finish a course and then allow the microorganisms to ‘change their colors’ and develop a resistance to the previously susceptible antibiotic,” Kevin E. Whitlock noted. “Microorganisms require complete extermination and when this does not happen, the few remaining organisms may become resistant.”

Dr. Adalja asserted laboratory professionals and other healthcare workers will have to remain vigilant for the presence of this bacteria and, when it is present, abide by strict infection control procedures including vigorously monitoring hand hygiene, donning appropriate gloves and gowns and ensuring proper antimicrobial therapy is initiated. He also stressed healthcare practitioners can decrease the impact of the bacterium simply by ensuring 100 percent compliance with established infection control guidelines.

**References**


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If you wish to submit answers manually (only available to AMT members), the fee is $10/test. Please submit a copy of this page with your answers marked, along with a completed order form located elsewhere in this publication. Don’t forget to include payment.

1. *A. baumannii* has origins in:
   - A. daycare populations
   - B. troops returning home from the Middle East
   - C. well water
   - D. veterinary clinics

2. *Acinetobactor baumannii* can be carried on the skin by normal healthy individuals without any clinical consequences.
   - A. True
   - B. False

3. *A. baumannii* is on the rise because of:
   - A. understaffed facilities, particularly healthcare practitioner to patient ratios.
   - B. inappropriate use of medical equipment.
   - C. the proportion of patients in hospitals that transition from hospital to nursing home and long-term care facility and back several times.
   - D. decreased antimicrobial resistance.

4. MRSA is a type of staph bacteria resistant to treatment with certain group of antibiotics including:
   - A. fluvastatin sodium.
   - B. acetaminophen and oxycodone.
   - C. lisinopril, moexipril, and perindopril.
   - D. methicillin, oxacillin, penicillin and amoxicillin.

5. *A. baumannii* infections principally occur in strong, irregularly hospitalized elderly individuals.
   - A. True
   - B. False

6. Which of the following is not typical of *A. baumannii?*
   - A. UTIs
   - B. wound infection
   - C. bloodstream infections
   - D. myocardial infarction

7. Multidrug resistant organisms have developed and present a new public health hazard related to, but not exclusively caused by:
   - A. extensive use of antibiotics.
   - B. increased allergies to latex gloves.
   - C. handwashing techniques.
   - D. compromised hosts.

8. Patients who do not complete a prescribed course of antibiotics typically pose a risk to their own healthcare.
   - A. True
   - B. False

9. Which of the following can best help curb the spread of multidrug resistant organisms?
   - A. innovative laboratory methods
   - B. vigorous hand hygiene
   - C. discontinued use of ventilators
   - D. patient discharge data

10. What approach can best help reduce the impact of *A. baumannii?*
    - A. increased use of antibiotics
    - B. increased staffing in long-term care facilities
    - C. ensuring 100 percent compliance with established infection control guidelines
    - D. frequent communication with the Centers for Disease Control and other reporting agencies
To submit STEP answers manually (only available to AMT members), you must complete this form and attach the appropriate quiz(es) with your payment and mail to American Medical Technologists.

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- Medical Assisting: Are You Doing What You are Supposed to be Doing?
- Medical Errors/HIPAA (HITECH Act)
- Clinical Microbiology-Case Studies
- D-Dimer and its Application in Suspected VTE
- Critical Thinking and Problem Solving for Medical Assistants
- Followership: You Choose Your Path
- PSA, Free PSA, Complex PSA and Prostate Cancer
- Vitamin D -- Why all the Fuss?
- Medical Errors in Healthcare Settings
- Dealing with Difficult People
- Update on Aspirin and Plavix Sensitivity and Resistance Testing
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Topics
- Professionalism in the Workplace
- IDEAL Communication: The Art of Conflict Resolution
- Improving Listening Skills
- AMT Certification Process (For schools and students)
Almost 30 years ago, Ivan Illich wrote a book on *Medical Nemesis*, which suggested that medical care can do more physical and social harm than good. He described it as “social iatrogenesis” suggesting that medical damage to an individual can occur by a sociopolitical mode. He suggested that medical bureaucracy creates ill-health by increasing stress, by multiplying disabling dependence, by generating new painful needs, by lowering the levels of tolerance for discomfort or pain and by abolishing even the right to self-care.

Three years ago, Shannon Brown wrote a book entitled “Overtreated: How much Medicine is Making Us Sicker and Poorer.” This impressed Dr. Vikai Saini, a Harvard cardiologist and president of The Lown Foundation, to set up a conference at Cambridge, Massachusetts, and invited 130 prominent doctors from USA, Canada and the UK. The purpose of the conference was to explore the problem of over-investigation and over-treatment of patients. U.S.A.’s per capita spending was rising sharply, faster than life expectancy. The conference concluded that the reasons for this ever increasing cost of medical care were due to:

1) Fear of malpractice lawsuits  
2) Supply driven demand  
3) Knowledge gaps  
4) Biased research  
5) Profit seeking  
6) Patient demand  
7) Financial conflicts of guideline writers  
8) Failure to inform the patient of potential harm  
9) The way US doctors are paid by fee of service.

Participants poured out examples of overuse of screening tests and imaging technology as well as an epidemic of doubtful benefit surgery. Tonsillectomies went up by 74% from 1996 to 2006. Cancer screening for the over-75-years age group and increasing rates of percutaneous coronary interventions also increased dramatically.

There was general agreement that guidelines should be written by those who are free of conflicts of interest, reduce hospital stay and reform tort law. There was some disagreement that the Affordable Care Act would reduce hospital costs.

Each culture has its own characteristic perception of disease and its preferred method of healing. Cost is irrelevant in achieving good health and longevity. Some doctors think that a patient refuses invasive treatment because he or she is in denial. In her work on palliative care, Diane Meier said it was not the patient who was in denial but the doctor.

Is the U.S.A’s Problem Unique?

Having worked on both sides of the pond, I feel I am able to judge the pros and cons of our medical industry. The UK is insulated against needless over-testing, over-treating and over-diagnosing. However, medical practice in the UK is governed by the National Institute for Health and Clinical Excellence (NICE), which offers evidence-based medical care. The National Screening Committee provides clinical recommendations of what screening is effective and useful. Medical care is governed by politics. The present coalition government in the UK intends to bring in “value based” medical care in 2014 rather than following the recommendations of NICE. Medical care in the UK is becoming too expensive and the NHS of which the UK prides itself may disintegrate into community health care units controlled by bureaucrats. The judge determines what is legal and who is guilty. The priest declares what is holy and who has sinned. The physician decides what is a symptom and who is sick. He is a moral entrepreneur, charged with inquisitorial powers to discover certain wrongs to
be righted. The medicalization of a budget is the share taken out of a person's yearly income to spend under doctor's orders.

**ObamaCare — Basic Facts**
The official name for “ObamaCare” is the Patient Protection and the Affordable Care Act. It is a bill that was signed on March 23, 2010, by President Barack Obama. It is intended to reform the health care industry. The basic facts are as follows:

1) The Affordable Care Act requires insurance plans to cover preventative services.
2) It stops insurance companies from dropping US citizens when they are sick.
3) It does not replace private insurance, Medicare or Medicaid.
4) It aims to improve community health care centers to provide improved health care for those who cannot afford private health care.
5) ObamaCare puts a cap on insurance companies raising premiums starting in 2013 and into 2014. The insurance companies will have to justify rate hikes to the state and post them to their websites.
6) Starting in October, 2013, insurance companies will compete via a health insurance exchange pool to decrease insurance premiums for all Americans.
7) Americans will buy affordable health care coverage on the “ObamaCare” health insurance exchanges.
8) American employers with 25 but less than 50 employees may receive tax breaks of up to 35% of the cost of their employees’ insurance premiums. Employers with more than 50 employees must insure their workers or pay a tax (like the current state run unemployment and workers compensation programs).
9) Only 3% of small businesses will have to pay the additional 0.9% ObamaCare Medicare tax increase. This tax is only paid on profitable income over $250K.
10) If the citizen chooses not to purchase healthcare through the Online Health Insurance Exchange, they can still buy private insurance, get insurance through work or Medicare/Medicaid. Those who choose to not participate will pay a penalty tax.
11) ObamaCare expects to cut $716 billion of waste from Medicare and cut reimbursements to private Medicare Advantage plans and reinvest it into Obama's health care reform.
12) 19 million Americans will receive tax credits to help pay for healthcare.
13) By 2019, 17 million Americans below the poverty line will be eligible for Medicaid due to expansion of the program.
14) Over 20.4 million women will gain access to new women's health care preventative services and better access to wellness visits and free preventative care on all insurance plans.

**Where is ObamaCare Heading?**

Whether one is a Republican or Democrat, the nation has voted that ObamaCare is here to stay. From a politician's perspective, this is a political dream come true. From a patient's point of view, this is nowhere near the "free for all" medical care of the British National Health Service system. Is the patient's autonomy safeguarded? Is there a balance of the needs and the demands of the patient? Can we afford the high standard of medical care we have in the USA without capping the payments made by the high malpractice awards? From the profession's perspective, does it limit the extensive investigation and treatments offered to patients and, therefore, reduce their income and threaten jobs of ancillary staff? The purpose of any health care is to restore function and maintain autonomy. The innovative practitioner should therefore develop new ways of maintaining a good patient-professional relationship without risking the patient's rights to healthcare and thus leading to social iatrogenesis.

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If you wish to submit answers manually (only available to AMT members), the fee is $10/test. Please submit a copy of this page with your answers marked, along with a completed order form located elsewhere in this publication. Don’t forget to include payment.

1. Which of these terms is NOT synonymous with MEDICAL NEMESIS?
   A. Social Iatrogenesis
   B. Medical Dependence
   C. Medical bureaucracy does not create ill health
   D. Overtreatment can cause more harm

2. Which of the following is NOT a cause of overtreatment?
   A. Fee for service medical care
   B. Supply driven demand
   C. People are more sick than they used to be
   D. Fear of malpractice lawsuits

3. Over treatment and over investigation is dependent on good insurance or ability to pay
   A. True
   B. False

4. Under the Affordable Care Act 2010 insurance companies will compete for health insurance by
   A. January 2013
   B. October 2013
   C. January 2014
   D. January 2015

5. Medicaid will be available for 17 million Americans below the poverty line by 2014
   A. True
   B. False

6. How will Obamacare affect MEDICARE?
   A. Increase by $455 billion
   B. Will be cut by $716 billion
   C. Would not be affected
   D. Will run out of funds by 2019

7. The free for all British National Health System is becoming very expensive
   A. True
   B. False

8. How many Americans will receive tax credits to help pay for health care?
   A. 8 Million
   B. 19 Million
   C. 24 Million
   D. 32 Million

9. What does the author think will NOT keep costs of medical health care down?
   A. Cap malpractice awards
   B. Follow strict guidelines of testing and treatment
   C. Agree to patient’s request for over investigation.
   D. Develop guidelines for necessary investigation and treatment

10. How does Obamacare affect women’s health
    A. Offer women less access to preventive health care
    B. Offer women less planned parenthood services
    C. Offer women less counseling for plastic surgery
    D. Offer women less access to better health care.
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As the people who perform a number, if not all, the glucose runs in our institutions, we have an important role to play in Tight Glycemic Control (TGC). Over the past several years, the idea of maintaining a patient’s glucose within a rather narrow range has been proposed and argued against. It appears that at least some, if not much, of the argument is the definition of TGC. This could be in large part due to the method for measuring glucose on which the limits for TGC are set. For example, if the lower limit is set at 110 or 120 mg/dL, this could be due to the instrument used and/or the sample — whole blood or serum.

Let’s consider some of the research that has been done on this topic and see how the laboratory can assist the clinicians who are dependent on us for providing the important data.

One of the early studies was done in Leuven, Belgium. “Leuven I” was the study that launched many of the current TGC protocols; in the ICU, TGC has been endorsed by a number of professional societies and is currently an emerging standard of care worldwide. Nevertheless, after nearly six years, there has been a lack of confirmatory evidence in the critical care literature, and conversely, a number of studies that argue against TGC due to higher mortality. In 2004, the 1,600-patient “before and after” study performed by an ICU team and reported as the Stamford (Connecticut) Study provided data confirming the benefit of TGC in a mixed medical-surgical population. “Leuven II,” undertaken in a medical ICU setting, emerged five years after the first Belgian study, detailing more modest benefits of intensive insulin therapy.

How does one explain the divergent results of different interventional trials regarding TGC and mortality? Leuven I and the Stamford studies were strongly positive; Leuven II was positive when considering only a targeted group of patients with ICU stays of longer than three days but negative in the intention-to-treat analysis of the entire group. Moreover, the results of two trials have cast more doubt about the efficacy of TGC. The European investigators of the Efficacy of Volume Substitution and Insulin Therapy in Severe Sepsis trial performed a multicenter, randomized, prospective study of intensive vs. “conventional” glycemic control and two different fluid resuscitation strategies in a two-by-two design. The trial was closed prematurely because of a nearly six-fold increase in severe hypoglycemia among the patients in the intensively treated group. The Glucontrol Trial, another multicenter European study of TGC in medical-surgical ICUs, was halted due to protocol violations and an unacceptable rate of hypoglycemia in the intensively treated group.

These two trials share one important parameter with Leuven II — a high rate of severe hypoglycemia (SH) [usually defined as blood glucose <40 mg/dL] and therefore might not be deemed indictments of TGC but rather as examples of failed trials. The rates of SH among the conventionally and intensively treated patients in Leuven II were 3.1% and 18.7%, respectively. In contrast, the corresponding rates in Leuven I were 0.8% and 5.1%, and the rate of SH in the Stamford study did not change with implementation of TGC. The Leuven II authors stated that “(statistical) analysis identified hypoglycemia as an independent risk factor for death. Hence, it is possible that hypoglycemia induced by intensive insulin therapy may have reduced a portion of the potential benefit.” Indeed, even a single episode of SH conferred an increased risk of mortality in an analysis of the 5,365 patient cohort from the Stamford study.

Why was the rate of hypoglycemia so different in the two Leuven studies? One important difference between Leuven I and Leuven II concerns the method of glycemic monitoring.
study relied exclusively on arterial blood analyzers, while the second study used arterial blood in an unspecified smaller number of patients, with the remainder of samples obtained from capillary blood and measured on bedside glucometers. Another difference is the patient population. The patients in the medical ICU had much higher APACHE (acute physiology and chronic health evaluation) II scores and a higher prevalence of sepsis on admission, risk factors for the development of SH, than did the surgical ICU patients.

Consider the role the laboratory plays in helping to achieve any TGC goal. In the case of blood glucose, four glucose measurement methods are used:

- hand-held devices (POCT)
- paper or plastic sticks to test a drop of blood
- blood gas analyzers
- laboratory analyzers in core laboratories

Each method is subject to specific challenges and limitations that can affect the overall system performance. Weber and Neeser point out several salient facts concerning the identification and documentation of the type of the sample. They argue that fasting glucose values in venous blood are 5–10% lower than in arterial samples, while capillary samples show 5–15% higher values compared to venous blood samples. Similarly, venous or capillary plasma samples are showing 10–15% higher values compared to whole blood hemolysate. As was pointed out at the beginning of this article, these difference can easily cause the variation in the low limit for glucose control. Lastly, as we are well-aware, hand-held instruments for measuring glucose have not always been found without significant error and variation.

Krinsley points out that the avoidance of hypoglycemia requires several key components. The ICU culture must accept protocol-driven care; a glycemic target should be chosen that can be achieved safely; the strengths and weaknesses of the different available glucose monitoring technologies must be understood; and finally, clinicians need to have access to actionable, real-time data, not just relating to glucose control, but ideally also to relevant outcomes such as severity-adjusted mortality and measures of morbidity and resource utilization.

References

6. stat.kuleuven.be/consulting/Show%20cases/LDP.pdf
Questions for STEP Participants

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If you wish to submit answers manually (only available to AMT members), the fee is $10/test. Please submit a copy of this page with your answers marked, along with a completed order form located elsewhere in this publication. Don’t forget to include payment.

1. What does TGC stand for?
   A. Today’s glucose calibrator
   B. Tight glycemic control
   C. Tight glycemic calibration
   D. Terrific glucose control

2. Where was one of the earliest studies of TGC performed?
   A. John’s Hopkins
   B. Leuven
   C. Loma Linda
   D. Los Alamos

3. TGC is maintained by employing which of the following therapies?
   A. Intensive glucose therapy
   B. Strict dietary requirements managed by a nutritionist
   C. Intensive insulin therapy
   D. Continuous glucose monitoring

4. What study confirmed the benefit of TGC in a medical-surgical population?
   A. Leuven I
   B. Leuven II
   C. The Stanford Study
   D. The Stamford (Connecticut Study)

5. What is the generally accepted definition of Severe Hypoglycemia?
   A. Blood Glucose <40 mg/dl
   B. Blood Glucose <50 mg/dl
   C. Blood Glucose <60 mg/dl
   D. Blood Glucose <70 mg/dL

6. What three trials all resulted in an unacceptably high rate of Severe Hypoglycemia in the studies’ patients?
   A. Leuven I, Leuven II and Stamford
   B. Efficacy of Volume Substitution and Insulin Therapy in Severe Sepsis trial, Leuven I and the Glucontrol trial
   C. Efficacy of Volume Substitution and Insulin Therapy in Severe Sepsis trial, the Glucontrol trial and Leuven II
   D. Efficacy of Volume Substitution and Insulin Therapy in Severe Sepsis trial, the Glucontrol and Stamford

7. What methodology did Leuven I use to monitor blood glucose in its patients?
   A. Fingerstick bedside glucometers
   B. Arterial blood gas analyzers
   C. Laboratory analyzers in core laboratories
   D. Plastic sticks to test blood drop

8. Which sample type yields a higher glucose value?
   A. Whole blood
   B. Plasma
   C. Urine
   D. Serum

9. What is the key point in avoiding hypoglycemia in the ICU culture?
   A. Accept protocol driven care
   B. A glycemic target should be chosen that can be achieved safely
   C. Strengths and weaknesses of the different available glucose monitoring technologies must be understood
   D. All of the above

10. TGC was found to result in more
    A. Normal glucose levels in more patients
    B. A greater hunger in patients
    C. A lesser hunger in patients
    D. Hypoglycemia in more patients
Thalassemias

There is a group of disorders which are all different and inherited and called thalassemias. Essentially, they are defined by a decreased or absent production of a specific globin chain.

Hemoglobin molecule

A hemoglobin molecule is made up of two alpha chains and two other chains, either beta, gamma, or delta. Normal adult hemoglobin is Hemoglobin A, consisting of two alpha chains and two beta chains. This is about 97% of normal hemoglobin. A small percentage of hemoglobin A2 and hemoglobin F is also present in the population. Hemoglobin F is predominately in fetuses and newborns.

The thalassemias are classified by a decrease or absent production of a specific globulin chain. Thus we see that beta thalassemia is the most common form of thalassemia and is identified by a decrease or absence of beta chains. Alpha thalassemia is a reduction or absence of alpha chains.

The inheritance of any type of thalassemia may be homozygous or heterozygous. Depending on the state of inheritance, the disorder is then known as beta thalassemia minor or thalassemia major.

References:

Submitted by Gerard P. Boe., Ph.D.

We welcome reader submissions for future “Fast Facts.” Send them to the AMT Office, attention Journal Editor.
Kingella kingae was initially isolated by Elizabeth O. King in 1960 during her tenure at the Center for Disease Control. She sent two strains to Bovre and Henriksen at the University of Oslo, Norway, asking if they thought the new isolate was a member of the genus Moraxella and, if so, if it was a member of an existing species. She died before the species could be fully described, but Kingella kingae is now recognized as an important pediatric pathogen. Improvements in culture and identification methods have led to Kingella kingae being detected more frequently. It is now known to be the leading cause of pediatric osteoarticular infections (OAI), infection of bone or joint, for which a causative agent has been determined.

Though a cause of endocarditis, bacteremia, and lower respiratory infection, Kingella kingae is most commonly seen as a causative agent of OAI. Its role in endocarditis led to Kingella kingae being included in the HACEK (Haemophilus, Aggregatibacter, Cardiobacterium, Eikenella, and Kingella) grouping of organisms. As Kingella’s growing importance is recognized, it is critical to understand the organism’s presentation, pathogenesis, epidemiology, identification, and treatment.

After further study, Bovre and Henriksen renamed the species Kingella kingae and placed it in the family Neisseriaceae, which also contains the genus Neisseria and the genus Eikenella. Three other species of the genus Kingella have since been discovered: Kingella oralis, Kingella denitrificans, and Kingella potus (Table 1). Analysis of 16S RNA sequencing suggests that Kingella kingae, Kingella oralis, and Kingella potus are very closely related, while Kingella denitrificans is more closely related to Eikenella corrodens.

Clinical Significance and Disease

Kingella kingae has been implicated in a variety of diseases, including osteoarticular infections (OAI), septic arthritis, lower respiratory tract infections, meningitis, endocarditis, and afocal bacteremia. The use of automated blood culture instruments for blood and other body fluids (like joint fluid, beginning in 1992) has increased the ability to detect Kingella kingae. This has led to an increased appreciation for the role of Kingella kingae in disease.

In a 2007 French study, Sylvia Chometon and colleagues found that Kingella kingae is now the leading cause of osteoarticular infections in children. This study used three different methods to detect Kingella kingae: automated blood culture machines to culture joint fluid, PCR with universal 16S primers on culture negative samples, and Kingella-specific PCR and sequencing on cultures negative after universal PCR. The use of these three techniques led to a high rate of Kingella kingae detection. Causative agents were determined for 86 of 131 samples. Of these, 39 (45%) were positive for Kingella kingae. Staphylococcus aureus, the most common causative agent when only automated culture results were considered, was found in only 29% of samples, even when S. aureus specific 16S sequencing was added.
A second study found that *K. kingae* is second only to *S. aureus* as the leading cause of OAI (33). 36.5% of positive cultures were positive for *S. aureus*, while 22.2% were positive for *K. kingae*. Only automated culture allowed detection of *K. kingae*. Unlike the previous study, only solid culture media and automated joint fluid culture were used to detect pathogens. It is likely that some cases of *K. kingae* were not identified, as no PCR was performed on culture negative samples. Taken together, these studies show that *K. kingae* is the leading cause of OAI and that automated blood culture machines are necessary to effectively identify *K. kingae* from joint fluid.

Though skeletal infections comprise most cases of invasive *K. kingae* infection, other disease presentations are also common. In one study of patients in an Israeli hospital, 69.4% of *K. kingae* infections were skeletal infections, 29.7% were bacteremia, and 4.1% were lower respiratory infections (36). All cases were in patients under four years of age, except for one adult who had *K. kingae* endocarditis. *K. kingae* endocarditis is relatively rare and typically occurs in children with pre-existing heart conditions (37).

The source of invasive *K. kingae* infections is likely colonization of the oropharynx (37). In one study, blood samples and throat swabs were collected from three patients with invasive *K. kingae* infections. *K. kingae* was isolated from all blood and throat specimens. Pulse field gel electrophoresis and random amplified polymorphic DNA PCR were performed on each isolate. For all three patients, the patterns seen for the blood isolate were identical to the throat isolate and distinct from other, non-related strains. This suggests that *K. kingae* involved in invasive infections originates from normal oropharyngeal colonization by *K. kingae*. Though colonization appears to be the source of invasive *K. kingae*, colonization is not considered a health risk. In healthy colonized children, the probability of developing *K. kingae* OAI is less than one percent (2).

Other *Kingella* species have also been implicated in disease. *K. denitrificans* has been identified as the causative agent in cases of infective endocarditis, both on native valves (11, 12, 26) and prosthetic valves (17). It has also been isolated from cases of granulomatous disease in an AIDS patient (21), amniotic fluid from a woman with chorioamnionitis (20), pus from empyema in a patient with lung cancer (22), and a retropharyngeal abscess in a patient on methotrexate (23). *K. oralis* is commonly found in dental plaque and other oral sites (3). It is unclear if *K. oralis* plays a pathogenic role or merely colonizes these areas. *K. potus* has been isolated as the

---

### Table 1. *Kingella* species

<table>
<thead>
<tr>
<th></th>
<th><em>K. kingae</em></th>
<th><em>K. denitrificans</em></th>
<th><em>K. oralis</em></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gram Stain</strong></td>
<td><img src="image1" alt="Image" /></td>
<td><img src="image2" alt="Image" /></td>
<td><img src="image3" alt="Image" /></td>
</tr>
<tr>
<td><strong>Growth on Sheep Blood Agar</strong></td>
<td><img src="image4" alt="Image" /></td>
<td><img src="image5" alt="Image" /></td>
<td><img src="image6" alt="Image" /></td>
</tr>
<tr>
<td><strong>Growth on Chocolate Agar</strong></td>
<td><img src="image7" alt="Image" /></td>
<td><img src="image8" alt="Image" /></td>
<td><img src="image9" alt="Image" /></td>
</tr>
</tbody>
</table>
causative agent of a wound infection from a bite from a kinkajou, a South American mammal (19).

Pathogenic Mechanisms

The process through which colonization of the oropharynx leads to invasive Kingella kingae infection is not fully understood. Several potential contributing factors have been proposed. In one study, 80% of patients with invasive K. kingae infections also had a concurrent respiratory or gastrointestinal illness. Due to the high frequency of co-infections, it has been hypothesized that viral infections disrupt the respiratory epithelium, allowing K. kingae to enter the bloodstream. From the bloodstream, K. kingae can reach other areas of infection, like joints and the endocardium.

Kingella kingae also produces a cytotoxic RTX toxin. In vitro studies have shown that the organism is cytotoxic to conjunctiva cells, type II pneumocytes, laryngeal cells, synovial cells, and macrophage-like cells, but only when the RTX toxin is present. It is possible that production of the RTX toxin allows K. kingae to disrupt the laryngeal epithelium and enter the bloodstream, causing invasive disease. The RTX toxin's cytotoxicity to synovial cells may also help explain how Kingella kingae causes septic arthritis.

The production of a type IV pilus allows adherence of K. kingae to respiratory epithelium, promoting colonization. In vitro assays show that pilus expression is required for K. kingae adherence to laryngeal cells, type II pneumocytes, and synovial cells. This suggests that pilus expression may be involved both in colonization of the oropharynx and infection of joint synovia in OAI. Pili expression has been shown to correspond to both site of isolation and colony morphology. Clinical isolates from the respiratory tract and bloodstream are usually piliated, and these colonies form either spreading/corroding or non-spreading/non-corroding colonies, but not domed colonies. In contrast, isolates from sites of invasive infection, like joint fluid and the endocardium, are less likely to have pili and are more likely to form domed colonies. This suggests that there is a selective disadvantage on the expression of pili in invasive infection, though this is not yet fully understood. K. dentrificans also produces a type IV pilus.

Epidemiology and Routes of Transmission

When considering the prevalence of Kingella kingae, it is necessary to consider both the prevalence of colonization and the incidence of invasive infection. Colonization rates vary with age. K. kingae is usually acquired beginning around six months of age, with colonization rates peaking between twelve and twenty-four months of age. Within this age group, 9-12% of children are colonized. K. kingae is transmitted by droplet transmission from colonized children. Children that are frequently around other children, like those who attend day care, have higher rates of colonization.

Similarly, rates of K. kingae invasive infection are highest in the first two years. One study of an Israeli pediatric hospital found the incidence of K. kingae invasive infection to be 21.2 cases per 100,000 population in the first year of life and 22.6 cases per 100,000 population in the second year. These rates then fell quickly with an incidence of 4.8 cases per 100,000 population in the third year and 1.4 cases per 100,000 population in the fourth year. Children under the age of four account for 98.6% of invasive infections. Infections occurred at an average rate of 12 cases per 100,000 emergency department visits and did not change significantly during the study period.

While rare, two outbreaks of Kingella kingae have been reported in the literature to date. One outbreak occurred in southern Israel, while the other occurred in Minnesota. Each outbreak involved a single day care center from which three children developed osteomyelitis. In the Minnesota outbreak, all three affected children were in the same daycare classroom. K. kingae carriage rates in this classroom were 45%, much higher than the average 9-12%. In the Israeli outbreak, the daycare center the affected children attended had a carriage rate of 33.3%. In both outbreaks, the strain isolated from the affected patients and carriers attending the same day care center had similar band patterns when undergoing pulse field gel electrophoresis. This suggests that the same strain was spread among the children harboring K. kingae. Furthermore, strains were isolated from other day care centers in the same areas. When subjected to pulse field gel electrophoresis, these strains had similar patterns within each day care center, but not between centers. This suggests that a unique strain had spread among each individual
day care center. It also may suggest that certain Kingella strains are more prone to invasive infections than others, as children in other day care centers did not develop osteomyelitis.

**Laboratory Identification**

Kingella are Gram-negative bacilli with flat ends in pairs or short chains, though they may resist decolorization or decolorize unevenly. They are non-motile, facultative anaerobic bacteria capable of fermenting glucose and maltose. They are oxidase positive, indole negative, urease negative, and catalase negative. Biochemical characteristics that identify Kingella from other members of family Neisseriaceae are summarized in Table 3. Table 2. Defined characteristics of Kingella from other Kingella species are summarized in Table 3.

Reliable detection of Kingella requires the use of automated blood culture machines. Joint fluid can be incubated in a BACTEC aerobic blood culture bottle or other similar bottle. When the pathogen is Kingella, automated blood culture machines recognize growth after 2-4 days of incubation. Positive culture bottles can then be subcultured onto blood or chocolate agar. Selective media can also be used. Thayer-Martin agar or media containing clindamycin or vancomycin may be helpful in isolating Kingella, as these agars select against other common oropharyngeal flora. Direct inoculation of solid media with synovial fluid during surgery has also been successful in isolating Kingella from patients with osteomyelitis.

Once isolated, Kingella can be identified using any of several common laboratory systems. Two commercially available tests, Remel RapID NH and API NH strips, use dehydrated substrates to perform biochemical tests on isolated organisms. These can be read after 4-5 hours of incubation.

**Table 2. Biochemical differentiation between Kingella kingae and other species in family Neisseriaceae**

<table>
<thead>
<tr>
<th>Morphology</th>
<th>Kingella kingae</th>
<th>Kingella denitrificans</th>
<th>Kingella oralis</th>
<th>Kingella palpebrae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Catalase</td>
<td>* - - + + + + ssp</td>
<td>+ + + + + + +</td>
<td>- - - - - - +</td>
<td>- - - - - - +</td>
</tr>
<tr>
<td>Oxidase</td>
<td>- - - - - -</td>
<td>+ + + + + + +</td>
<td>- - - - - - +</td>
<td>- - - - - - +</td>
</tr>
<tr>
<td>Nitrite to Nitrate</td>
<td>- - - - - -</td>
<td>- - - - - - -</td>
<td>+ + + + + + +</td>
<td>- - - - - - +</td>
</tr>
<tr>
<td>Alkaline Phosphatase</td>
<td>- - - - - -</td>
<td>- - - - - - -</td>
<td>- - - - - - -</td>
<td>- - - - - - -</td>
</tr>
<tr>
<td>Ornithine</td>
<td>- - - - - -</td>
<td>- - - - - - -</td>
<td>- - - - - - -</td>
<td>- - - - - - -</td>
</tr>
<tr>
<td>Acid dehydrogenase</td>
<td>- - - - - -</td>
<td>- - - - - - -</td>
<td>- - - - - - -</td>
<td>- - - - - - -</td>
</tr>
<tr>
<td>Acid production from Glucose</td>
<td>- - - - - -</td>
<td>- - - - - - -</td>
<td>- - - - - - -</td>
<td>- - - - - - -</td>
</tr>
<tr>
<td>Maltose</td>
<td>- - - - - -</td>
<td>+ + + + + + +</td>
<td>- - - - - - +</td>
<td>- - - - - - +</td>
</tr>
<tr>
<td>Lactose</td>
<td>- - - - - -</td>
<td>- - - - - - -</td>
<td>+ + + + + + +</td>
<td>- - - - - - +</td>
</tr>
<tr>
<td>Sucrose</td>
<td>- - - - - -</td>
<td>- - - - - - -</td>
<td>+ variable + ssp</td>
<td>- - - - - - +</td>
</tr>
<tr>
<td>Fructose</td>
<td>- - - - - -</td>
<td>- - - - - - -</td>
<td>+ + + + + + +</td>
<td>- - - - - - +</td>
</tr>
</tbody>
</table>


+ more than 90% of strains positive; - more than 90% of strains negative; Ssp: some subspecies positive; blank: no info published

**Table 3. Biochemical differentiation between Kingella kingae and other species in genus Kingella**

<table>
<thead>
<tr>
<th>Biochemical Test</th>
<th>Kingella kingae</th>
<th>Kingella denitrificans</th>
<th>Kingella oralis</th>
<th>Kingella palpebrae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrite to Nitrate</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Alkaline Phosphatase</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Acid production from Glucose</td>
<td>+</td>
<td>+</td>
<td>weak</td>
<td>-</td>
</tr>
<tr>
<td>Maltose</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Lactose</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Sucrose</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>


+ more than 90% of strains positive; - more than 90% of strains negative
Automated systems may also be used to identify *Kingella kingae*. VITEK 2 NH identification cards and the VITEK 2 system use similar colormetric tests to identify *K. kingae*, though varying levels of accuracy have been reported\(^4\). Additionally, matrix-assisted laser desorption/ionization (MALDI) time of flight (TOF) mass spectrometry (MS) has recently been successfully used to identify *Kingella kingae*, Powell, manuscript in preparation).

Culture-independent methods have also been developed to identify *K. kingae*, especially from culture-negative samples\(^5\). Both universal and specific PCR have been employed. Universal PCR involves amplifying 16S ribosomal genes, sequencing the PCR product, and searching a database of known sequences to identify the bacteria from which the gene was isolated. Specific PCR involves the use of primers that amplify 16S ribosomal genes of *K. kingae* only. If any product results from the PCR, the sample is positive for *K. kingae*. Both methods have improved the detection rate for *K. kingae* in culture negative samples.

### Antibiotic Susceptibility

*K. kingae* is generally susceptible to beta-lactams and cephlosporins\(^7,10\). These drugs are most commonly used to treat invasive *K. kingae* infections. Several studies have also found them to be susceptible to aminoglycosides, macrolides, chloramphenicol, fluoroquinolones, tetracyclines, colistin, and rifampin. Occasional resistance has been seen to gentamicin, amikacin, trimethoprim-sulfamethoxazole, tobramycin, and oxacillin. Resistance patterns previously reported in literature are summarized in Table 4.

Recent testing in our laboratory on patient isolates, laboratory stock strains, and ATCC strain 23331 showed similar results. Antibiotic susceptibility testing was performed using E-test strips on cation-adjusted Mueller-Hinton agar with 5% sheep blood. Results were compared to standards published. Results are summarized in Table 5. All isolates were susceptible to ampicillin, ceftriaxone, ciprofloxacin, and meropenem. Four of 13 isolates were resistant to trimethoprim-sulfamethoxazole. Clindamycin was tested, and all isolates had minimum inhibitory drug concentrations between 1.5 and 16 μg/mL (Mean: 4.61 μg/mL). Standards have not been established for minimum inhibitory concentration interpretation of clindamycin with *K. kingae*, but relatively high minimum inhibitory concentrations suggest clindamycin may not be appropriate for use against *K. kingae*. Similar resistance patterns were seen with *K. denitrificans* and *K. oralis*, though only one isolate of each was tested.

### Conclusion

*Kingella kingae* is now recognized as an emerging fastidious pediatric pathogen, although it is likely still under-detected. The advent of blood culture instruments for culturing synovial fluid has led to increased rates of detection, but many cases likely still go undetected when PCR is not employed\(^6\). Development of standard *K. kingae* specific PCR assays would increase the detection rate of *K. kingae* and decrease the number of cases of OAI for which no cause can be determined. It is still unclear how *K. kingae* moves from the oropharynx to the bloodstream, but there is evidence for a role for the RTX toxin and viral co-infection. Further research may further elucidate this process, identifying potential targets for decreasing the incidence of systemic *K. kingae* infection.
Table 5. Antibiotic susceptibility in *Kingella* species isolates

<table>
<thead>
<tr>
<th></th>
<th>MIC50</th>
<th>MIC90</th>
<th>Range</th>
<th>% Susceptible</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>0.064</td>
<td>0.5</td>
<td>≤0.016-0.5</td>
<td>100</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>0.094</td>
<td>0.125</td>
<td>0.032-0.125</td>
<td>100</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>0.047</td>
<td>0.064</td>
<td>0.016-0.064</td>
<td>100</td>
</tr>
<tr>
<td>Meropenem</td>
<td>0.064</td>
<td>0.125</td>
<td>0.008-0.125</td>
<td>100</td>
</tr>
<tr>
<td>Trimethoprim-sulfamethoxazole</td>
<td>0.02</td>
<td>&gt;32</td>
<td>≤0.02-&gt;32</td>
<td>69.2</td>
</tr>
</tbody>
</table>

All values in μg/mL.

References

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1. *Kingella kingae* is most often associated with which of the following clinical diseases?
   A. Infectious endocarditis
   B. Osteoarticular infections
   C. Pneumonia
   D. Bacteremia

2. Which of the following is the most likely source for invasive *K. kingae* infections?
   A. Oropharyngeal colonization
   B. Skin lesions over joints
   C. Exposure to contaminated blood products
   D. Airborne bacteria

3. In which age group are *K. kingae* infections most common?
   A. 0-4 years
   B. 4-10 years
   C. 10-18 years
   D. adults

4. Which characteristic of *K. kingae* is INCORRECT?
   A. Gram-negative
   B. Oxidase positive
   C. Indole negative
   D. Catalase positive

5. Which technology has been successfully applied to the identification of *K. kingae* in the routine clinical laboratory?
   A. VITEK 2
   B. 16S sequencing
   C. MALDI-TOF
   D. All of the above

6. *K. kingae* is in what family?
   A. *Pasterellaceae*
   B. *Neisseriaceae*
   C. *Cardiobacteriaceae*
   D. *Enterobacteriaceae*

7. In addition to *K. kingae*, which of the following lists include the other species in the genus Kingella?
   A. *K. oralis, K. potus* and *K. denitrificans*
   B. *K. corrodans, K. potus* and *K. denitrificans*
   C. *K. mitis, K. proteus* and *K. denitrificans*
   D. *K. oralis, K. potus* and *K. decarboxylatus*

8. What risk factor has been identified for invasive *K. kingae* infection?
   A. Concurrent respiratory or GI infections
   B. Extended stay in the hospital
   C. Exposure to infected water
   D. Antibiotic treatment

9. In what population have *K. kingae* outbreaks been observed?
   A. Returning veterans of foreign wars
   B. Chronically ill children
   C. Children attending daycare
   D. No outbreaks have been observed.

10. To which class of antibiotics has resistance not been seen in *K. kingae*?
    A. Aminoglycosides
    B. Sulfonamides
    C. Beta-lactams
    D. Cephalosporins

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   B. Skin lesions over joints
   C. Exposure to contaminated blood products
   D. Airborne bacteria

3. In which age group are *K. kingae* infections most common?
   A. 0-4 years
   B. 4-10 years
   C. 10-18 years
   D. adults

4. Which characteristic of *K. kingae* is INCORRECT?
   A. Gram-negative
   B. Oxidase positive
   C. Indole negative
   D. Catalase positive

5. Which technology has been successfully applied to the identification of *K. kingae* in the routine clinical laboratory?
   A. VITEK 2
   B. 16S sequencing
   C. MALDI-TOF
   D. All of the above

6. *K. kingae* is in what family?
   A. *Pasterellaceae*
   B. *Neisseriaceae*
   C. *Cardiobacteriaceae*
   D. *Enterobacteriaceae*

7. In addition to *K. kingae*, which of the following lists include the other species in the genus Kingella?
   A. *K. oralis, K. potus* and *K. denitrificans*
   B. *K. corrodans, K. potus* and *K. denitrificans*
   C. *K. mitis, K. proteus* and *K. denitrificans*
   D. *K. oralis, K. potus* and *K. decarboxylatus*

8. What risk factor has been identified for invasive *K. kingae* infection?
   A. Concurrent respiratory or GI infections
   B. Extended stay in the hospital
   C. Exposure to infected water
   D. Antibiotic treatment

9. In what population have *K. kingae* outbreaks been observed?
   A. Returning veterans of foreign wars
   B. Chronically ill children
   C. Children attending daycare
   D. No outbreaks have been observed.

10. To which class of antibiotics has resistance not been seen in *K. kingae*?
    A. Aminoglycosides
    B. Sulfonamides
    C. Beta-lactams
    D. Cephalosporins
In an article entitled “Mononuclear Leukocytosis in Reaction to Acute Infection (infectious mononucleosis),” which appeared in the Bulletin of the John Hopkins Hospital in 1920, the authors described the clinical characteristics of the causative organism of the disease.

Since the 1800s, infectious mononucleosis had been recognized as a clinical syndrome of fever, adenopathy and pharyngitis. But it wasn’t until the late 1960s that the association between infectious mononucleosis and the Epstein-Barr Virus (EBV) was described.

Infectious Mononucleosis is also called Pfeiffer’s Disease and Filatov’s Disease named for two researchers who described the syndrome as an infectious process, the first by Filatov in 1887, and later, independently by Pfeiffer in 1889.

It is easily transmitted orally and is so often called the “kissing disease.” It is said to be more common among adolescents and young adults. The Epstein-Barr virus (EBV) is a member of the Herpes virus family which is said to infect almost everyone at some point in their lives. EBV often causes no symptoms but it can cause mononucleosis.

**Classic Symptoms**

The very basic symptoms are sore throat, fatigue, pharyngeal inflammation, vomiting, loss of appetite and petechiae. Infectious mononucleosis occurs with the viral infection of Epstein-Barr virus. The virus is spread by saliva and has an incubation period of from four to seven weeks. The symptoms usually persist for two to three weeks but the feeling of malaise and fatigue may last longer. The most commonly used diagnostic criterion is the presence of 50% or more lymphocytes with at least 10% atypical lymphocytes (large, irregular nuclei). The atypical lymphocytes resembled monocytes when they were first discovered, and thereafter the term mononucleosis, was used.

**General Description of Leukocytes in Infectious Mononucleosis**

In infectious mononucleosis, the total white cell count is variable, but it is usually increased over the normal. There is a relative and an absolute increase in the cells of the lymphocytic series. Usually this lymphocytosis is greater than 60 percent.

The feature of this lymphocytosis is the variability of the cells as compared with the uniformity of the cell types in leukemia.

Neutrophils are relatively decreased with a slight shift to the left. The eosinophils, however, tend to be slightly increased while Basophils, typical lymphocytes and typical monocytes are also present.

**Case Studies**

# 1 - Peripheral blood (Wright’s stain)

A teen-aged Caucasian boy was in good health when he suddenly developed a sore throat, fever, and generalized enlargement of the lymph nodes. Three days after the onset of the illness, the WBC count was 10,000, with 85 percent lymphocytes. One week later, the WBC count was 37,000 with 85 percent lymphocytes, 3 percent monocytes, 11 percent segmented neutrophils and 1 percent eosinophils.

**Clinical Diagnosis: Infectious mononucleosis**

Photos contain atypical lymphocytes in infectious mononucleosis. The cytoplasm of the larger...
cell is blue and contains small red granules. Red blood cells indent the margins of the larger cell.

#2 Peripheral blood (Wright’s stain)

This 3-yr old boy reported with generalized enlargement of the lymph nodes and spleen.

His WBC count was 19,000, with 72 per cent lymphocytes.

Clinical Diagnosis: Infectious mononucleosis.

Photos show the cytoplasm of the cell is bluish gray and granular and has a small but definite vacule. The nucleus has “brain-like” convolutions.

#3 Peripheral blood (Wright’s stain)

This 20 year old white male had a severe sore throat and a low grade fever. The mucous membrane of his nasopharynx was inflamed and hemorrhagic. His cervical lymph nodes were enlarged.

Clinical Diagnosis: Infectious mononucleosis.

Photos show small and large lymphocyte and monocyte in infectious mononucleosis. Also shown are a monocyte and atypical early cell of infectious mononucleosis.

#4 Peripheral blood (Wright’s stain)

The initial findings in this 30-year old man were fever, headache, sore throat, and enlargement of lymph nodes. The patient was in bed for approximately ten days, and for one month thereafter, he felt weak. His heterophile agglutination titer was 1:128 and his WBC count was 12,500.

Photos show lymphocytes with multiple chromophobic areas in their cytoplasm. Notice the variability of the atypical cells versus the uniformity of the normal cells.

Differential Diagnosis

There are two disorders in patients with primary infection which should be closely evaluated versus IM. They are acute cytomegalovirus infection and Toxoplasma gondii infections. Because the clinical signs of the above mentioned tests are very similar, it is often difficult to tell between EBV mono and the cytomegalovirus infection. It is not always helpful, or possible, to distinguish between EBV mononucleosis, but in pregnant women, the test should be completed because of the consequences for the fetus.

In patients with a primary EBV IM infection, the demonstration of heterophile is diagnostic. One point of information here is the fact that the original Paul-Bunnell test has been replaced with rapid qualitative agglutination or ELISA tests. The tests detect 80-85% of IM. A negative test should simply imply the absence of significant IM-specific heterophile antibodies. Heterophile antibodies appear in 60% of patients with IM within the first and second weeks, and in 80-90% by the first month.

References


Murray PR, Baron, EJ, Jorgensen, JH et.al, editors: Manual of clinical microbiology, Washington, DC 20003, American Society for Microbiology. Wu, Alan HB, editor

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1. The connection of the Epstein-Barr virus to infectious mononucleosis was described as early as 1887.
   A. True
   B. False

2. EBV has an incubation period from four to seven weeks.
   A. True
   B. False

3. One of the most commonly used diagnostic criteria is the Presence of 50% or more of lymphocytes.
   A. True
   B. False

4. It is often difficult to identify IM cells because there is little variability when compared to the uniformity of cell types in Leukemia.
   A. True
   B. False

5. Heterophile antibodies are not necessarily diagnostic of EBV mononucleosis.
   A. True
   B. False

6. EB mononucleosis is called “the kissing virus” because so many teenagers get it.
   A. True
   B. False

7. Sore throat, fatigue, and pharyngeal inflammation are considered some of the basic symptoms.
   A. True
   B. False

8. The lymphocytosis in this disease is usually greater than 60% .
   A. True
   B. False

9. There is an absolute as well as relative increase in lymphocytes in the disease.
   A. True
   B. False

10. Atypical lymphocytes are primarily identified by their large, irregular nuclei.
    A. True
    B. False
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Since its isolation, Methicillin-resistant Staphylococcus aureus (MRSA) has become a major cause of hospital-acquired infection (HAI), adverse patient outcome and overall resource utilization. This is a retrospective study of the rates and outcomes of MRSA infection in orthopedic trauma at the Royal Infirmary of Edinburgh. This study found significant associations between adverse patient outcome (persistent deep infection, osteomyelitis, the necessity for revision surgery, amputation and mortality) and the following patient variables: Length of inpatient stay, immuno-compromise, pre-admission residence in an institutional setting (such as a residential nursing home) and the number of antibiotics used in patient care. Despite 63% of all infections sampled resulting from proximal femoral fractures, no association between patient outcome and site of infection or diagnosis was found. Somewhat surprisingly, the relationship between age and outcome of infection was not proved to be significant, contradicting previous studies suggesting a statistical association. Antibiotic prophylaxis, previously identified as a factor in reducing overall incidence of MRSA infection, was not found to be significantly associated with outcome. Early identification of high-risk patients as identified by this study could lead to more judicious use of therapeutic antibiotics and reductions in adverse outcome, as well as socioeconomic cost. These results could assist in more accurate risk stratification based on a evidence based evaluation of the significance of the risk factors investigated.


During the past two decades, methicillin-resistant Staphylococcus aureus (MRSA) has become increasingly common as a source of nosocomial infections. Most studies of MRSA surveillance were performed during outbreaks, so that results are not applicable to settings in which MRSA is endemic. This paper gives an overview of MRSA prevalence in hospitals and other healthcare institutions in non-outbreak situations in Western Europe. Thirty-one observational studies were included in the review. Four of the studies were of good quality. Surveillance screening of MRSA was performed in long-term care (11 studies) and acute care (20 studies). Prevalence rates varied over a wide range, from less than 1% to greater than 20%. Prevalence in the acute care and long-term care settings was comparable. The prevalence of MRSA was expressed in various ways -- the percentage of MRSA among patients (range between 1% and 24%), the percentage of MRSA among Staphylococcus aureus isolates (range between 5% and 54%), and as the prevalence density (range between 0.4 and 4 MRSA cases per 1,000 patient days). The screening policy differed with respect to time points (on admission or during hospital stay), selection criteria (all admissions or patients at high risk for MRSA) and anatomical sampling sites. This review underlines the methodological differences between studies of MRSA surveillance. For comparisons between different healthcare settings, surveillance methods and outcome calculations should be standardized.


Owing to a high prevalence of methicillin-resistant Staphylococcus aureus (MRSA) among residents, long-term-care facilities (LTCFs) have become substantial reservoirs of this microorganism. Few data on the natural history of MRSA colonization in this setting are available. The cumulative incidence appears to be approximately 20% per year, and more than half of carriers have persistent colonization. Several host-related factors -- such as antibiotic use, invasive devices, and poor infection control practices -- increase the risk of colonization. Clinical experience suggests that subsequent MRSA infections are neither frequent nor severe while colonized residents are living in an LTCF; however, when admitted to an acute-care center, Colonized individuals may spread MRSA to other patients and may develop severe infections. Therefore, the epidemiological impact of the high prevalence of MRSA in LTCFs is less relevant than the clinical impact of this colonization for an individual resident. Standard precautions should be applied as routine infection control measures for all residents of LTCFs, whereas barrier precautions, cohorting, decolonization and other measures should be undertaken only for controlling outbreaks of MRSA infection.
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