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Universal vaccination of children 6 to 59 months of age with trivalent inactivated influenza vaccine has recently been recommended by U.S. advisory bodies. To evaluate alternative vaccine approaches, we compared the safety and efficacy of intranasally administered live attenuated influenza vaccine with those of inactivated vaccine in infants and young children. Children 6 to 59 months of age, without a recent episode of wheezing illness or severe asthma, were randomly assigned in a 1:1 ratio to receive either cold-adapted trivalent live attenuated influenza vaccine with those of inactivated vaccine in infants

ORIGINAL ARTICLES


In early 2005, physicians at a rural hospital in KwaZulu-Natal, a province of South Africa, were concerned by a high rate of rapid death among patients infected with the human immunodeficiency virus (HIV) who also had tuberculosis. A study revealed the presence not only of multidrug-resistant (MDR) tuberculosis but also what came to be called extensively drug-resistant (XDR) tuberculosis. XDR tuberculosis is caused by a strain of Mycobacterium tuberculosis resistant to isoniazid and rifampin (which defines MDR tuberculosis) in addition to any fluoroquinolone and at least one of the three following injectable drugs: capreomycin, kanamycin, and amikacin. Of 53 patients with XDR tuberculosis, 55% claimed they...

For patients with chronic pancreatitis and a dilated pancreatic duct, ductal decompression is recommended. We conducted a randomized trial to compare endoscopic and surgical drainage of the pancreatic duct. All symptomatic patients with chronic pancreatitis and a dilated pancreatic duct but without an inflammatory mass were eligible for the study. We randomly assigned patients to undergo endoscopic transampullary drainage of the pancreatic duct or operative pancreaticojejunostomy. The primary end point was the average Izbicki pain score during 2 years of follow-up. The secondary end points were pain relief at the end of follow-up, physical and mental health, morbidity, mortality, length of hospital stay, number of procedures undergone, and changes in pancreatic function. Thirty-nine patients underwent randomization: 19 to endoscopic treatment (16 of whom underwent lithotripsy) and 20 to operative pancreaticojejunostomy. During the 24 months of follow-up, patients who underwent surgery, as compared with those who were treated endoscopically, had lower Izbicki pain scores (25 vs. 51, P<0.001) and better physical health summary scores on the Medical Outcomes Study 36-Item Short-Form General Health Survey questionnaire (P=0.003). At the end of follow-up, complete or partial pain relief was achieved in 32% of patients assigned to endoscopic drainage as compared with 75% of patients assigned to surgical drainage (P=0.007). Rates of complications, length of hospital stay, and changes in pancreatic function were similar in the two treatment groups, but patients receiving endoscopic treatment required more Surgical drainage of the pancreatic duct was more effective than endoscopic treatment in patients with obstruction of the pancreatic duct due to chronic pancreatitis.


Long-acting beta-agonists and inhaled corticosteroids are used to treat chronic obstructive pulmonary disease (COPD), but their effect on survival is unknown. We conducted a randomized, double-blind trial comparing salmeterol at a dose of 50 µg plus fluticasone propionate at a dose of 500 µg twice daily (combination regimen), administered with a single inhaler, with placebo, salmeterol alone, or fluticasone propionate alone for a period of 3 years. The primary outcome was death from any cause for the comparison between the combination regimen and placebo; the frequency of exacerbations, health status, and spirometric values were also assessed. Of 6112 patients in the efficacy population, 875 died within 3 years after the start of the study treatment. All-cause mortality rates were 12.6% in the combination-therapy group, 15.2% in the placebo group, 13.5% in the salmeterol group, and 16.0% in the fluticasone group. The hazard ratio for death in the combination-therapy group, as compared with the placebo group, was 0.825 (95% confidence interval [CI], 0.681 to 1.002; P=0.052, adjusted for the interim analyses), corresponding to a difference of 2.6 percentage points or a reduction in the risk of death of 17.5%. The mortality rate for salmeterol alone or fluticasone propionate alone did not differ significantly from that for placebo. As compared with placebo, the combination regimen reduced the annual rate of exacerbations from 1.13 to 0.85 and improved health status and spirometric values (P<0.001 for all comparisons with placebo). There was no difference in the incidence of ocular or bone side effects. The probability of having pneumonia reported as an adverse event was higher among patients receiving medications containing fluticasone propionate (19.6% in the combination-therapy group and 18.3% in the fluticasone group) than in the placebo group (12.3%, P<0.001 for comparisons between these treatments and placebo). The reduction in death from all causes among patients with COPD in the combination-therapy group did not reach the predetermined level of statistical significance. There were significant benefits in all other outcomes among these patients.


The Björnstad syndrome, an autosomal recessive disorder associated with sensorineural hearing loss and pili torti, is caused by mutation of a previously unidentified gene on chromosome 2q34–36.efined genetic mapping and DNA sequencing of 44 genes between D2S2210 and D2S2244 revealed BCS1L mutations. Functional analyses elucidated how BCS1L mutations cause the Björnstad syndrome. BCS1L encodes a member of the AAA family of ATPases that is necessary for the assembly of complex III in the mitochondria. In addition to the Björnstad syndrome, BCS1L mutations cause complex III deficiency and the GRACILE syndrome, which in neonates are lethal conditions that have multisystem and neurologic manifestations typifying severe mitochondrial disorders. Patients with the Björnstad syndrome have mutations that alter residues involved in protein–protein interactions, whereas mutations in patients with complex III deficiency alter ATP-binding residues, as deduced from the crystal structure of a related AAA-family ATPase. Biochemical studies provided evidence to support this model: complex III deficiency mutations prevented ATP-dependent assembly of BCS1L-associated complexes. All mutant BCS1L proteins disrupted the assembly of complex III, reduced the activity of the mitochondrial electron-transport chain, and increased the production of reactive oxygen species. However, only mutations associated with complex III deficiency increased mitochondrial content, which further increased the production of reactive oxygen species. BCS1L mutations cause disease phenotypes ranging from highly restricted pili torti and sensorineural hearing loss (the Björnstad syndrome) to profound multisysytem organ failure (complex III deficiency and the GRACILE syndrome). All BCS1L mutations disrupted the assembly of mitochondrial respirasomes (the basic unit for respiration in human mitochondria), but the clinical expression of the mutations was correlated with the production of reactive oxygen species. Mutations that cause the Björnstad syndrome illustrate the exquisite sensitivity of ear and hair tissues to mitochondrial dysfunction, particularly to the production of reactive oxygen species.


Persons who have hypermethylation of one allele of MLH1 in somatic cells throughout the body (a germ-line epimutation) have a predisposition for the development of cancer in a pattern typical of hereditary nonpolyposis colorectal cancer. By studying the families of two such persons, we found evidence that the epimutation was transmitted from a mother to her son but was erased in his spermatozoa. The affected maternal allele was inherited by three
other siblings from these two families, but in those offspring the allele had reverted to the normal active state. These findings demonstrate a novel pattern of inheritance of cancer susceptibility and are consistent with transgenerational epigenetic inheritance.


Epidemiologic data suggest that infection with herpes simplex virus type 2 (HSV-2) is associated with increased genital shedding of human immunodeficiency virus type 1 (HIV-1) RNA and HIV-1 transmissibility. We conducted a randomized, double-blind, placebo-controlled trial of HSV suppressive therapy with valacyclovir (at a dose of 500 mg twice daily) in Burkina Faso among women who were seropositive for HIV-1 and HSV-2; all were ineligible for highly active antiretroviral therapy. The patients were followed for 24 weeks (12 weeks before and 12 weeks after randomization). Regression models were used to assess the effect of valacyclovir on the presence and quantity of genital and plasma HIV-1 RNA and genital HSV-2 DNA during treatment, adjusting for baseline values, and to evaluate the effect over time. A total of 140 women were randomly assigned to treatment groups; 136 were included in the analyses. At enrollment, the median CD4 cell count was 446 cells per cubic millimeter, and the mean plasma viral load was 4.44 log10 copies per milliliter. With the use of summary-measures analysis, valacyclovir therapy was found to be associated with a significant decrease in the frequency of genital HIV-1 RNA (odds ratio, 0.41; 95% confidence interval [CI], 0.21 to 0.80) and in the mean quantity of the virus (log10 copies per milliliter, −0.29; 95% CI, −0.44 to −0.15). However, there was no significant decrease in detection of HIV (risk ratio, 0.93; 95% CI, 0.81 to 1.07). HSV suppressive therapy also reduced the mean plasma HIV-1 RNA level.


Treatment of acute Kawasaki disease with intravenous immune globulin and aspirin reduces the risk of coronary-artery abnormalities and systemic inflammation, but despite intravenous immune globulin therapy, coronary-artery abnormalities develop in some children. Studies have suggested that primary corticosteroid therapy might be beneficial and that adverse events are infrequent with short-term use. We conducted a multicenter, randomized, double-blind, placebo-controlled trial to determine whether the addition of intravenous methylprednisolone to conventional primary therapy for Kawasaki disease reduces the risk of coronary-artery abnormalities. Patients with 10 or fewer days of fever were randomly assigned to receive intravenous methylprednisolone, 30 mg per kilogram of body weight (101 patients), or placebo (98 patients). All patients then received conventional therapy with intravenous immune globulin, 2 g per kilogram, as well as aspirin, 80 to 100 mg per kilogram per day until they were afebrile for 48 hours and 3 to 5 mg per kilogram per day thereafter. At week 1 and week 5 after randomization, patients in the two study groups had similar coronary dimensions, expressed as z scores adjusted for body-surface area, absolute dimensions, and changes in dimensions. As compared with patients receiving placebo, patients receiving intravenous methylprednisolone had a somewhat shorter initial period of hospitalization (P=0.05) and, at week 1, a lower erythrocyte sedimentation rate (P=0.02) and a tendency toward a lower C-reactive protein level (P=0.07). However, the two groups had similar numbers of days spent in the hospital, numbers of days of fever, rates of retreatment with intravenous immune globulin, and numbers of adverse events...


RRM1, the regulatory subunit of ribonucleotide reductase, is involved in carcinogenesis, tumor progression, and the response of non–small-cell lung cancer to treatment. We developed an automated quantitative determination of the RRM1 protein in routinely processed histologic specimens. In these specimens, we measured the expression of RRM1 and two other proteins that are relevant to non–small-cell lung cancer: the excision repair cross-complementation group 1 (ERCC1) protein and the phosphatase and tensin homologue (PTEN). We compared the results with the clinical outcomes in 187 patients with early-stage non–small-cell lung cancer who had received only surgical treatment. RRM1 expression correlated with the expression of ERCC1 (P<0.001) but not with the expression of PTEN (P=0.37). The median disease-free survival exceeded 120 months in the group of patients with tumors that had high expression of RRM1 and was 54.5 months in the group with low expression of RRM1 (hazard ratio for disease progression or death in the high-expression group, 0.46; P=0.004). The overall survival was more than 120 months for patients with tumors with high expression of RRM1 and 60.2 months for those with low expression of RRM1 (hazard ratio for death, 0.61; P=0.02). Among these 187 patients, the survival advantage was limited to the 30% of patients with tumors that had a high expression of both RRM1 and RRM1 and ERCC1 are determinants of survival after surgical treatment of early-stage, non–small-cell lung cancer.

CLINICAL PRACTICE


This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author’s clinical recommendations. A 36-year-old man with a 20-year history of type 1 diabetes mellitus, background retinopathy, peripheral sensory neuropathy, and nephropathy presents with a history of several months of nausea and vomiting of undigested food and bile, during which time he lost 4 kg. On physical examination (performed 1 hour after the patient has eaten), his blood pressure is 130/80 mm Hg while he . . .

REVIEW ARTICLE


The link between myocardial ischemia and obstructive atherosclerosis of the epicardial coronary arteries is well established, and coronary angiography has demonstrated a relationship between the severity and extent of coronary artery disease (CAD) and survival. In the past two decades, however, a number of studies have reported that abnormalities in the function and structure of the coronary microcirculation occur in many clinical conditions. In some instances these abnormalities represent epiphenomena, whereas in others they
represent important markers of risk or may even contribute to the pathogenesis of myocardial ischemia, thus becoming therapeutic targets.


Stroke is one of the most feared complications of surgery. To provide adequate preventive and therapeutic measures, physicians need to be knowledgeable about the risk factors for stroke during the perioperative period. In this article, I review the pathophysiology of perioperative stroke and provide recommendations for the stratification of risk and the management of risk factors. The incidence of perioperative stroke depends on the type and complexity of the surgical procedure. The risk of stroke after general, noncardiac procedures is very low. Cardiac and vascular surgeries — in particular, combined cardiac procedures — are associated with higher risks.

**IMAGES IN CLINICAL MEDICINE**

(Since these articles has no abstract, we just provided an extract of the first 100 words of the full text and any section headings)


A 76-year-old man with hypertension presented with a sudden, painless, and profound loss of vision in the right eye. Visual acuity was light perception only in the affected eye, with a positive relative afferent papillary defect. Ophthalmoscopic examination revealed diffuse retinal whitening, constriction of the arteriole and venule with segmentation (Panel A, arrowhead), and a cherry-red spot in the macula (Panel A, arrow) — all signs compatible with the diagnosis of central retinal artery occlusion. A horizontally oriented optical coherence tomograph of the macula (Panel B, arrowhead) demonstrated increased thickness and reflectivity of the inner bands, indicating ischemic damage to . . .


A 64-year-old man presented with progressive shortness of breath and exercise intolerance due to end-stage ischemic cardiomyopathy. Since he remained severely symptomatic despite maximal medical therapy, he underwent a heterotopic cardiac transplantation. Because of the presence of severe pulmonary hypertension, the recipient’s native heart (N) was left in place and the allograft was implanted in the right chest. The native heart maintains right circulation in spite of chronic pulmonary hypertension, while the heterotopic donor heart (D) functions as a biologic left ventricular assist device. The post-transplantation electrocardiogram shows two QRS complexes with different axes (Panel A). The allograft can be seen clearly in the right chest on both the radiograph (Panel B) and the computed tomographic scan (Panel C) of the chest. An automatic implantable cardiac defibrillator and cardiac medications are used to treat the recipient’s native heart, as are immunosuppressive medications for the allograft.


An afebrile 8-year-old Ethiopian girl presented with a limp. Two years earlier, she had had mild trauma followed by “bone infection” and had received short courses of oral antibiotics. Examination revealed a small, pus-secreting wound on the anterior aspect of her left thigh. Her blood count was normal, but her erythrocyte sedimentation rate was 48 mm in the first hour. A radiograph of both legs (Panel A) and an axial computed tomographic scan (Panel B) with reformattting in the sagittal plane (Panel C) showed severe deformity of the left femur, proximal small lytic lesions, destruction and condensation throughout with sequestration (white arrows in Panels A and C and black arrows in Panel B), involucrum formation (arrowheads in all three panels), atrophy of the quadriceps muscles, and a posterolateral sinus (white arrow in Panel B). On open biopsy of the left femur, copious pus was drained. Pathological evaluation showed active, chronic osteomyelitis, with gram-positive cocci and involucrum (a sheath of new bone surrounding the sequestrum [necrosed bone]). A culture grew methicillin-sensitive Staphylococcus aureus and Streptococcus pyogenes. After drainage and partial resection of the sequestrum, the girl was treated with parenteral antibiotics and underwent orthopedic procedures.


54-year-old man presented to the emergency room with a 1-week history of influenza-like symptoms. He had undergone renal transplantation 12 years earlier for diabetic nephropathy. Laboratory evaluations were notable for an increase in his serum creatinine level to 6.0 mg per deciliter (530 µmol per liter) and 3+ proteinuria. Because of worsening renal failure, a renal biopsy was performed, which showed moderate chronic allograft nephropathy. In addition, an incidental finding of a heart-shaped, attenuated proximal renal tubule was seen. The influenza-like symptoms resolved within 2 weeks; however, the renal failure progressed, and the patient is once again being treated . . .

**CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL**

(Since these articles has no abstract, we just provided an extract of the first 100 words of the full text and any section headings)


Presentation of Case: A 28-year-old man was referred to this hospital for consultation on the management of an enlarging testicular mass. One year earlier, he had noticed a small, nontender mass in the posterior aspect of the right testicle, which a physician, who was a relative of the patient, ascribed to epididymitis; the mass seemed to disappear, or at least the patient did not notice it again. One month before the consultation, the results of a routine annual physical examination were normal; no abnormalities were noted in the testicles. Ten days before the consultation, the right testicle became tender and began to enlarge . . .


A 53-year-old man with a prosthetic aortic valve was admitted to this hospital because of the recent onset of fatigue, dyspnea, weight loss, and sweats. Approximately 4 years earlier, severe aortic insufficiency had developed. Echocardiography revealed a calcified, bicuspid aortic valve. Aortic-valve replacement was performed elsewhere, with a Medtronic Hall tilting-disk valve. Three months later, aortic insufficiency recurred, and the aortic valve was replaced with another Medtronic Hall valve at the same hospital. During the second operation, there was partial dehiscence of the prosthesis along a portion of the annulus, and although there was no abscess, inflammatory tissue was present at the surgical site. Pathological examination of the excised tissue disclosed a foreign-body giant-cell reaction; no organisms were identified.

**EDITORIALS**

(Since these articles has no abstract, we just provided an extract of the first 100 words of the full text and any section headings)
Corey, L. (2007). Synergistic copathogens: HIV-1 and HSV-2. New England Journal of Medicine, 356 (8) 854-856. The variability in both the clinical progression and transmission of human immunodeficiency virus (HIV) infection has prompted a search for cofactors influencing replication of the virus. Although it is clear that host immune and genetic factors, as well as the replication kinetics of particular viral strains, influence the progression of HIV disease, a variety of exogenously acquired infectious agents also appear to influence the pace of HIV replication, the destruction of CD4+ T cells, and HIV transmission to infants and sexual partners. Transient bursts of HIV replication occur after vaccination and during episodes of acute systemic infection. More persistent elevations in plasma HIV levels have been seen in patients with chronic infections (such as those with Mycobacterium tuberculosis and herpes and hepatitis viruses), and such coinfected patients have a more rapid loss of CD4+ T cells and an increased rate of progression to AIDS and death.

Cox, N.J., and Carolyn Buxton Bridges. (2007). Inactivated and live attenuated influenza vaccines in young children: how do they compare? New England Journal of Medicine, 356 (7), 727-731. Two quite different influenza vaccines are now licensed for use in the United States—a trivalent inactivated influenza vaccine that is administered intramuscularly and a live attenuated influenza vaccine that is administered by means of a nasal spray. The trivalent inactivated vaccine was first licensed for use in military populations during the 1940s. Recommendations for its use have expanded over the years to include groups at high risk for complications from influenza infection and their close contacts, including health care personnel, and recently also to include young children from 6 to 59 months of age and household contacts.

Elta, G.H. (2007). Is there a role for the endoscopic treatment of pain from chronic pancreatitis? New England Journal of Medicine, 356 (7), 727-729. The treatment of the pain of chronic pancreatitis continues to be problematic. Although severe calcific pancreatitis can be asymptomatic, it can cause disabling pain daily. Medical management with abstinence from alcohol, a low-fat diet, supplementary pancreatic enzymes, and nonnarcotic analgesic agents is the first step in therapy but is often ineffective. Pain management with the long-term use of narcotics is common but is often unsatisfactory because of tolerance to the medication, loss of efficacy, and complications of drug dependency. Injections of octreotide are used for pain control, but efficacy studies have been contradictory. Notably, the placebo response rate in patients.

Gosden, R.G., and Andrew P. Feinberg. (2007). Genetics and epigenetics: nature's pen-and-pencil set. New England Journal of Medicine, 356 (7), 731-733. The sequence of the four nucleotides of the genetic code is like an indelible ink that, with rare exceptions, is faithfully transcribed from cell to cell and from generation to generation. But on top of this code lies another one, literally “epigenetic,” which is represented by methyl groups added to the DNA base cytosine, as well as covalent changes in histone proteins around which the DNA is coiled. This epigenetic information is more like a code written in pencil in the margins around the DNA. Although the genome largely distinguishes one person from another, the epigenome, or epigenetic information, distinguishes . . .

Irby, D.M. (2007). Educational continuity in clinical clerkships. New England Journal of Medicine, 356 (8) 856-857. Continuity in medical-student clerkships is becoming a thing of the past. There is little continuity between students and teachers, between students and patients, and between specialty-based components of the curriculum. Although block rotations in clerkships have been used for more than 100 years, in Abraham Flexner’s day, patients, teachers, and students were together in the hospital for extended periods on medicine, obstetrics, and surgery services, which provided excellent opportunities to learn in a relatively relaxed and longitudinally mentored environment. Not so today.

Rabe, K.F. (2007). Treating COPD: the TORCH trial, P values, and the dodo. New England Journal of Medicine, 356 (8) 851-854. In the United States, the overall, age-standardized death rate decreased from 1242 deaths per 100,000 population in 1970 to 845 deaths per 100,000 in 2002. This good news must be viewed against the doubling during the same interval of the age-standardized death rate among persons with chronic obstructive pulmonary disease (COPD), 1 which makes COPD a major cause of death (Figure 1).

HEALTH POLICY REPORT

Iglehart, J.K. (2007). Medicaid revisited: skirmishes over a vast public enterprise. New England Journal of Medicine, 356 (7), 734-740. Medicaid, the federal–state program that provides protection against the costs of acute and chronic illness for almost one of every five Americans, has once again come under closer scrutiny from health policymakers. The greater attention paid to Medicaid, usually a neglected stepchild as compared with Medicare, has derived from an effort by Republicans to scale back federal spending on domestic activities and a strong plea by governors to reduce the growth of Medicaid because they regard it as unsustainable in its current form. From 1995 to 2005, total (federal and state) expenditures for Medicaid increased from $144.9 billion to $315.2 . . .

CLINICAL IMPLICATIONS OF BASIC RESEARCH

Staudt, L.M. (2007). A closer look at follicular lymphoma. New England Journal of Medicine, 357(7), 741-742. A majority of healthy persons have, in their normal B cells, the defining event of follicular lymphoma—a t(14;18) translocation that brings together BCL2 and the gene encoding the immunoglobulin heavy chain (Figure 1). The expression of BCL2 (which inhibits apoptosis) is consequently increased and contributes to the transformation of the normal B cell into follicular lymphoma. These events alone are not enough for complete transformation; additional processes are required. A recent report by Roulland et al.1 describes one such process and indicates that the cells with t(14;18) in healthy persons are closer to follicular lymphoma . . .