DOWNLOAD PDF PT. 1. PANEL DISCUSSION WITH EDITORS ET AL.

Chapter 1 : Reports | EUBlockchain

23rd Wealth Creation Study panel discussion with Mr. Raamdeo Agrawal Mr. Ramesh Damani, Mr. Akash Prakash moderated by Latha Venkatesh on CNBC TV

Half of these have delayed testicular descent, with the testis in the scrotum by weeks after term. Beyond this spontaneous descent is rare. Current treatment recommendations are that UDT beyond 3 months need surgery between months of age. Some children have scrotal testes in infancy but develop UDT later in childhood because the spermatic cord does not elongate with age, leaving the testis behind as the scrotum moves further from the groin. Many authors recommend surgery once the testis no longer resides spontaneously in the scrotum, but some groups recommend conservative treatment. Insulin-like hormone 3 INSL3 from developing Leydig cells stimulates the genito-inquinal ligament, or gubernaculum, to enlarge. This holds the testis near the inguinal canal as the fetal abdomen enlarges. Androgens guide this complex process, both directly and indirectly via a neurotransmitter, calcitonin gene-related peptide CGRP , released from the genitofemoral nerve. After migration is complete the proximal processus vaginalis closes preventing inguinal hernia and then disappears, allowing the spermatic cord to elongate with age, to keep the testis scrotal. Anomalies of the complex inguinoscrotal phase account for most UDT seen clinically. The maldescended testis suffers heat stress when not at the lower scrotal temperature 33 degrees Celsius , interfering with testicular physiology and development of germ cells into spermatogonia. UDT interrupts transformation of neonatal gonocytes into type-A spermatogonia, the putative spermatogenic stem cells at months of age. Recent evidence suggests orchidopexy between months improves germ cell development, with early reports of improved fertility, but no evidence yet for changes in malignancy prognosis. If this process is disrupted hypospadias occurs, with a variable proximal urethral meatus, failed ventral preputial development producing a dorsal hood, and discrepancy in the ventral versus dorsal penile length, causing a ventral bend in the penis, known as chordee. Surgery to correct hypospadias is recommended between months, as technical advances now allow operation to be done before long-term memory of the surgery. For complete coverage of this and related aspects of Endocrinology, please visit our FREE web-book, www. Not only is it prevalent, but also there remain unresolved questions about prognosis in adult life. However, current treatment is based on the assumption that early surgery will prevent germ cell degeneration during childhood, leading to improved fertility and fewer tumors 5 , 6. Our understanding of the embryology has advanced rapidly in recent years, with new theories and experimental evidence supporting a complex anatomical process controlled directly and indirectly by hormones 7 , 8. The classification of cryptorchidism also is changing, with the recent recognition of acquired anomalies 9 , With so much change in the way we view and treat cryptorchidism, endocrinologists will need to keep checking on the evolving controversies described in this chapter. EMBRYOLOGY The testes descend prenatally from their initial intra-abdominal location on the urogenital ridge into the low-temperature environment of the scrotum via a complex multi-stage mechanism Prior to weeks of development, the gonadal position is similar in both sexes. During the initial transabdominal phase of descent, regression of the cranial ligament and thickening of the gubernaculum allows the testis to be held near the inguinal region By contrast, in the female the cranial ligament persists while the gubernaculum remains thin and elongates, which together hold the ovary higher on the posterior abdominal wall as the fetal abdomen enlarges. The inguinal canal forms by the abdominal wall muscles developing around the caudal, gelatinous end of the gubernaculum, which initially ends at the future external inguinal ring. By 15 weeks the testis is attached by a short, stout and gelatinous gubernaculum to the future internal inguinal ring, while the ovary is higher in the pelvis In mid gestation a diverticulum of the peritoneal membrane, known as the processus vaginalis, begins to elongate within the gubernaculum, which retains a central connection known as the gubernacular cord with the epididymal tail and the lower pole of the testis. The caudal end of the gubernaculum grows out of the abdominal wall and elongates towards the scrotum, extending the processus vaginalis eventually to the scrotum. After the testis reaches the scrotum, two further anatomical events complete the inguinoscrotal phase, the first of which is obliteration of the proximal processus vaginalis The second event is involution of the

gelatinous gubernacular bulb and its anchoring to the inside of the scrotum. The former process prevents inguinal hernia or hydrocele and the latter process prevents extravaginal or perinatal torsion of the testis 6. The embryological stages of testicular descent and the postnatal growth required to keep the testis in the scrotum. The two main phases of descent appear to be controlled independently by hormones. INSL3 is the major factor controlling gubernacular enlargement and androgen, particularly DHT, and AMH appear to play minor roles in this "swelling reaction" of the gubernaculum Under the influence of the hormones mentioned above, the caudal end of the gubernaculum, where it attaches to the inguinal abdominal wall, enlarges by proliferation of the embryonic mesenchyme and deposition of extracellular matrix. Androgens also are responsible for regression of the cranial suspensory ligament, but they are not sufficient alone for transabdominal descent. The phase of gubernacular migration is controlled both directly and indirectly by androgens, with the aid of the genitofemoral nerve GFN releasing calcitonin gene-related peptide CGRP 23 , Androgens act during a critical time window to regulate gubernacular development Recent evidence suggests that the androgen receptors controlling this masculinization of the GFN may not be in the nerve itself, but in the target organ, the inguinoscrotal fat pad in the mammary line The number of sensory neurons and the amount of CGRP in the genitofemoral nerve of rats are significantly less after exposure to the anti-androgen, flutamide, consistent with androgens stimulating structural and functional changes in the nerve. The nerve is proposed to orient the direction of gubernacular migration, while the physical force needed for elongation of the processus vaginalis is probably provided by intra-abdominal pressure CGRP released from the nerve stimulates mitosis and cremaster muscle development in the gubernacular tip, enabling elongation to the scrotum Estrogens have a minor inhibitory role in normal gubernacular development, but estrogenic endocrine disruptors may be responsible in larger doses for cryptorchidism secondary to suppression of the "swelling reaction" by inhibition of INSL3. The trigger that initiates active migration of the caudal tip of the gubernaculum may come from the inguinoscrotal fat pad in the mammary line 28 , as the androgen receptors are present in the mammary line mesenchyme but not in the adjacent gubernaculum during the critical window of androgenic programming in rodents 25 , The gubernaculum has a surprisingly close link with the embryonic breast in normal marsupials as well as in eutherian animal models, such as the rat and mouse, especially after they have been exposed to the antiandrogen, flutamide 30 , The primitive mammary line is in continuity with the apical ectodermal ridges of the upper and lower limb buds, and hence is likely to contain similar activated signaling systems as seen in limb bud development These signals are likely to initiate outgrowth of the gubernaculum from the abdominal wall, so that it can migrate to the scrotum. Hormonal defects in INSL3, AMH or androgenic action are identified only rarely, suggesting that mechanical anomalies may be more common. Those patients with hormonal defects may present with rare disorders of sexual development DSD with cryptorchidism as part of the complex genital anomaly. As the gubernacular swelling reaction holds the testis close to the inguinal canal while other structures grow further away, the transabdominal phase is only relative movement of the testis and hence less likely to be abnormal. By contrast, the inguinoscrotal migration phase requires very significant mechanical and anatomical re-arrangements, and consequently, anomalies are common: Transient deficiency of androgen production in utero, perhaps related to deficiency of gonadotropin production by the fetal pituitary or the placenta 34 , may account for some, particularly where there is intra-uterine growth retardation. Anomalies of the genitofemoral nerve also may cause undescended testes. For example, perineal testes may be caused by an anomalous location of the genitofemoral nerve Inherited syndromes frequently are associated with cryptorchidism. Hypothalamic dysfunction, connective tissue disorders, neurogenic e. Cryptorchidism is also common in infants with abdominal wall defects, such as exomphalos or omphalocele, gastroschisis and exstrophy of the bladder There is much current interest in the potential adverse effects of environmental estrogenic endocrine disruptors on the incidence of both cryptorchidism and hypospadias In addition, there are data on the effect of diethylstilbestrol DES on cryptorchidism in male offspring of exposed mothers In the latter case there is supporting evidence from animal models 42 , although in the former, the cause-and-effect relationship is more tenuous, because the level of exposure is less clear, and the epidemiology may not have allowed for changes in diagnostic criteria over recent decades. More work is needed before we can ascertain a proven cause-and-effect link with synthetic

molecules in the environment. The body of the epididymis is hypoplastic and frequently is not tightly adherent to the cryptorchid testis This is more common in high intra-abdominal testes and probably indicates significantly decreased androgen production. Whether epididymal-testicular separation is the cause or the result of cryptorchidism is not known In addition, its effect on fertility is uncertain, even though the rete testis is nearly always still connected to the head of the epididymis. Recent studies show a strong link between maternal smoking and cryptorchidism in male offspring 45 , Beyond 12 weeks, spontaneous testicular descent is rare An undescended testis is best defined as a testis that cannot be manipulated into the bottom of the scrotum without excess tension on the spermatic cord by 12 weeks of age. A few cryptorchid testes are within the inguinal canal, making them impalpable unless they can be squeezed out of the external inguinal ring by compression. Ten percent of testes are intra-abdominal or absent presumed to be secondary to prenatal torsion. Whether this is a sign of primary endocrinopathy or secondary dysfunction of the testis, caused by heat stress when the gonad is not in the low temperature environment of the scrotum, is unknown. Postnatal increase in testosterone production is also diminished in premature infants, perhaps secondary to inadequate stimulation by chorionic gonadotropin in utero HCG is low compared with early pregnancy and may be of functional significance. Despite lower than normal androgen levels between 1 and 4 months of age, there is no apparent anomaly in androgen receptors from gonadal or skin biopsies collected at orchidopexy The postnatal secretion of both AMH and inhibin-B in cryptorchid infants is also deranged. Production of AMH from Sertoli cells normally increases between months, but this surge is blunted in undescended testes 55 , Inhibin-B normally increases at minipuberty and remains elevated into the second year of life , but levels in infants with cryptorchidism are lower This process is perturbed in cryptorchid testes, with failure of transformation of gonocytes into type-A spermatogonia between months These observations suggest that germ cell deficiency may be at least partly secondary to early postnatal dysfunction, rather than being congenital, as proposed by some authors 61 , Lack of germ cell transformation has been proposed to be secondary to postnatal androgen deficiency 60 , 63 or low AMH levels Recent studies, however, suggest that transformation is normal in both infants and mice with complete androgen insensitivity syndrome CAIS , and may be mediated by activin or another TGF-family factor Abnormal postnatal maturation of gonocytes could lead to both infertility and malignancy 65 , although some authors propose that there may be congenital carcinoma in-situ-cells in the cryptorchid testis 61 , 66 , There is now a consensus that type-A spermatogonia are likely to be the stem cells for future spermatogenesis, and that their appearance between 3 and 12 months of age, as neonatal gonocytes transform, is the key step in postnatal germ cell development 68 , Should this be confirmed, it implies that early surgical intervention should lead to an excellent prognosis, as long as the subsequent germ cell deficiency is secondary to postnatal heat stress of the maldescended testis, and therefore reversible. Failure of the totipotential gonocytes to transform into unipotential spermatogenic stem cells may leave some persisting gonocytes in the undescended testis, which is speculated to be the origin of subsequent tumors. DIAGNOSIS The aim of clinical examination is to locate the gonad, if palpable, and determine its lowest position without causing painful traction on the spermatic cord which probably corresponds to the caudal limit of the tunica vaginalis In infants, the diagnosis is straightforward because the scrotum is thin and pendulous. Hypoplasia of the hemiscrotum indicates it does not contain a testis. The inguinal testis is within its tunica vaginalis which gives it significant mobility. Ultrasonography has become more frequently used for diagnosis of the impalpable testis, but generally is not contributory for true intra-abdominal testes. This is because absence of the testis secondary to possible perinatal torsion is common, and also because intra-abdominal testes are often concealed by the bowel and other viscera In addition, the mobility of the undescended testis within its tunica vaginalis may make location by ultrasonography difficult. An ultrasound scan can be justified in bilateral impalpable testes, to confirm the presence of a testis. In addition AMH and inhibin-B should be measured to confirm the presence of functioning Sertoli cells A simple and reliable approach is to use laparoscopy, which readily locates the testis itself or blind-ending gonadal vessels , and allows orchidopexy in experienced hands TREATMENT Newborn and Infant Hormone therapy has become extremely controversial 73 , 74 as it was based on the two assumptions that cryptorchidism is not only secondary to a deficiency of the hypothalamic-pituitary-gonadal axis, but also the mechanical processes were simple. Randomized,

double-blind, placebo-controlled studies have not shown more than marginal benefit with either hCG or GnRH Despite proven endocrine control of descent, the mechanical factors appear to be too complex for this simple approach to be successful except for acquired undescended testes

Chapter 2 : WhenDisasterStrikesPt1

During the discussion, each panelist will read their poetry or prose or display their art. Book signing will follow with all contributors participating. About the editors: Wade Hudson is an author and president and CEO of Just Us Books, Inc., an independent publisher of books for children and young adults.

Content, goal orientation, and applicability should be reviewed on a regular basis. Training and informational components of PR should be delivered in a systematic manner to assure that all patient care issues are addressed. There should be repetition sufficient to ensure retention of information and skills. Giving the patient too much information at one time may cause confusion. Easy-to-read patient education materials should be used to complement and reinforce verbal instructions. Staff members should receive the influenza vaccination. Specific procedures are provided in the update of static lung volume measurement Section Instructions should be provided and techniques described in a manner that take into consideration the learning ability and communications skills of the patient being served. This Guideline does not apply. This Guideline is appropriate for children with indications who can be motivated and who can follow directions. This Guideline is appropriate for members of the geriatric population with indications who are motivated and who can follow directions. Pulmonary Rehabilitation Guideline Committee The principal author is listed first: Official statement of the American Thoracic Society. American Association of Cardiovascular and Pulmonary Rehabilitation. Guidelines for pulmonary rehabilitation programs, 2nd ed. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. Pulmonary rehabilitation in patients with COPD. Effects of pulmonary rehabilitation on physiologic and psychosocial outcomes in patients with chronic obstructive pulmonary disease. Ann Intern Med ; 1: Disease management of COPD with pulmonary rehabilitation. The long-term effects of pulmonary rehabilitation in patients with asthma and chronic obstructive pulmonary disease: Arch Phys Med Rehabil ;80 1: Foster S, Thomas HM 3rd. Pulmonary rehabilitation in lung disease other than chronic obstructive pulmonary disease. Am Rev Respir Dis ; 3: Outcomes and problems in pediatric pulmonary rehabilitation. Am J Phys Med Rehabil ;74 4: Exercise conditioning and cardiopulmonary fitness in cystic fibrosis: Effect of a home exercise training program in patients with cystic fibrosis. Bach JR Pulmonary rehabilitation in neuromuscular disorders. Pulmonary rehabilitation with respiratory complications of postpolio syndrome. Rehabil Nurs ;20 1: Pulmonary rehabilitation in chronic respiratory insufficiency. Psychiatric, psychosocial, and rehabilitative aspects of lung transplantation. Clin Chest Med ;11 2: Bilateral pneumonectomy volume reduction for chronic obstructive pulmonary disease. J Thorac Cardiovasc Surg ; 1: Analysis of chronic obstructive pulmonary disease referrals for lung volume reduction surgery. J Cardiopulm Rehabil ;17 4: Bernhard J, Ganz PA. Psychosocial issues in lung cancer patients Part I. Rehabilitation for the patient with advanced lung disease: Semin Respir Crit Care Med ; Psychological outcomes of a pulmonary rehabilitation program. Changes in self-concept during pulmonary rehabilitation, Parts 1 and 2. Heart Lung ;19 5 Pt 1: Definitions of health and health goals of participants in a community-based pulmonary rehabilitation program. Public Health Nurs ;10 1: Site and nature of airways obstruction in chronic obstructive lung disease. N Engl J Med ; The morphologic features of the bronchi, bronchioles, and alveoli in chronic airway obstruction: Am Rev Respir Dis ; 1: Pathophysiology of chronic obstructive pulmonary disease. Clin Chest Med ;11 3: Accuracy of two ear oximeters at rest and during exercise in pulmonary patients. Timed walking tests of exercise capacity in chronic cardiopulmonary illness. J Cardiopulm Rehabil ;16 1: The role of exercise testing in pulmonary diagnosis. Clin Chest Med ;8 1: Clinical exercise testing, 4th ed. Principles of exercise testing and interpretation, 3rd ed. American Association for Respiratory Care. Respir Care ; 36 Respir care ; 36 Respir Care ;38 5: Physical reconditioning of patients with respiratory diseases: Respir Care ;39 5: Upper extremity exercise training in chronic obstructive pulmonary disease. Supported arm exercise vs unsupported arm exercise in the rehabilitation of patients with severe chronic airflow obstruction. Pulmonary rehabilitation that includes arm exercise reduces metabolic and ventilatory requirements for simple arm elevation. Upper-limb and lower-limb exercise training in patients with chronic airflow obstruction. Chest ; 97 5: The effects of a 9-week program of aerobic and upper body

exercise on the maximal voluntary ventilation of chronic obstructive pulmonary disease patients. J Cardiopulm Rehabil ;15 2: Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness: JAMA ; 4: Rashbaum I, Whyte N. Occupational therapy in pulmonary rehabilitation: Collaborative self-management strategies for patients with respiratory disease. Respir Care ; 39 5: Outbreak of multiresistant non-encapsulated Haemophilus influenzae infections in a pulmonary rehabilitation centre. Pneumococcal polysaccharide vaccine efficacy: Sense and nonsense of influenza vaccination in asthma and chronic obstructive pulmonary disease. Geriatr Nurs ;18 2: Sexuality in the pulmonary patient. Prevalence and characteristics of nutritional depletion in patients with stable COPD eligible for pulmonary rehabilitation. Am Rev Respir Dis ; 5: Nutritional status and mortality in chronic obstructive pulmonary disease. Weight loss is a reversible factor in the prognosis of chronic obstructive pulmonary disease. Nutritional intervention in malnourished patients with emphysema. Am Rev Respir Dis ; 4: A comparison of sustained-release bupropion and placebo for smoking cessation. Meta-analysis on efficacy of nicotine replacement therapies in smoking cessation. Treating tobacco use and dependence. Also published in Respir Care ;45 Adherence in cardiac and pulmonary rehabilitation. J Cardiopulm Rehabil ;15 6: Discharge planning for the respiratory care patient. Respir Care ;40

Chapter 3 : Cryptorchidism and Hypospadias - Endotext - NCBI Bookshelf

Editor's Discussion Panel: When Disaster Strikes in the Editing Room! October 13, - PART 1. The Editors' Lounge Discussion Panel held on Friday the 13th of October , tackled the thorny problem of dealing with the setbacks that can occur in the editing suite and strategies to overcome them.

Hereditary neuropathies can manifest as a combination of sensory and motor neuropathy, isolated motor neuropathy or isolated sensory neuropathy sometimes with autonomic neuropathy. Charcot-Marie-Tooth disease Charcot-Marie-Tooth CMT disease is a group of hereditary neuropathies characterized by progressive muscle weakness and sensory loss in the arms and legs. Individuals in the early stages of the disease often present with clumsiness due to numbness in the feet. As the disease progresses, the lack of nerve conduction to the extremities can also result in depressed tendon reflexes, muscle atrophyâ€”especially at the ankles and hands, and foot deformities such as high, arched feet or hammertoes. Symptoms are caused by the impairment of the ability of peripheral nerves to conduct signals throughout the body, which results in reduced motor control and sensation in the arms and legs, especially at the ankles and wrists. Different subtypes of CMT are caused by different types of peripheral nerve abnormalities: Nerve conduction studies can be used in combination with inheritance pattern to determine the type of CMT CMT1, 2, 4, X, or dominant intermediate , but genetic testing is needed to identify the specific subtype e. Hereditary motor neuropathies Hereditary motor neuropathies HMNs , in some cases referred to as spinal muscular atrophies SMAs , are a clinically and genetically heterogeneous group of disorders characterized by loss of motor neurons within the spinal cord, resulting in weakness and muscle wasting. Typical clinical findings include slowly progressive muscle weakness and wasting. Onset of symptoms varies from the prenatal period to adulthood. Some forms of HMN also have minor involvement of the sensory neurons. Other features are variable depending on the causative gene, and may include vocal cord paralysis, facial weakness, pyramidal signs, and arthrogryposis. Many genes associated with HMN can also cause other forms of neuropathy with overlapping symptoms, such as Charcot-Marie-Tooth disease. Hereditary sensory and autonomic neuropathies Hereditary sensory and autonomic neuropathies HSAN are a clinically heterogeneous group of disorders that predominantly affect the sensory neurons of the peripheral nervous system, with or without autonomic neuron involvement. Loss of sensory neuron function can lead to complications including frequent injuries, ulcerations, bone infections and amputation. Possible autonomic features include anhidrosis, hyperhidrosis, abnormal blood pressure fluctuations, and gastrointestinal issues. Sensory abnormalities are often more significant than autonomic abnormalities. Motor neuron involvement may also occur in some individuals. Depending on the causative gene, other findings may also be present, such as hearing loss, gait impairment, decreased tendon reflexes, hypotonia, delayed development, and congenital insensitivity to pain. Age of onset varies from infancy to adulthood. Small fiber neuropathy Small fiber neuropathy SFNP is characterized by neuropathic burning or stabbing pain that typically occurs in the distal lower extremities and presents between adolescence and adulthood. Affected individuals typically experience heightened sensitivity to pain in general, but cannot feel pain concentrated in very small areas, such as a pinprick. Individuals may also have other sensory issues, such as the reduced ability to differentiate between hot and cold temperatures. Symptoms of this painful neuropathy may worsen over time and extend to include other parts of the body in addition to the hands and feet. The principal nerve biopsy finding consists of decreased small nerve fiber diameter. Riboflavin transporter deficiency neuronopathy Riboflavin transporter deficiency neuronopathy is a neurodegenerative disorder characterized by progressive axonal sensorimotor neuropathy. Clinical features of this disorder include bulbar palsy facial weakness, drooping eyelids, difficulty speaking and swallowing , weakness and distal muscle atrophy in the limbs typically more severe in the upper than lower limbs , respiratory distress due to diaphragmatic weakness, and gait ataxia. Sensorineural hearing loss occurs in many individuals and may be the presenting symptom. Historically, the clinical subtype of riboflavin transporter deficiency neuronopathy that includes hearing loss has been called Brown-Vialetto-Van Laere syndrome, and the clinical subtype that does not include hearing loss has been called Fazio-Londe disease. Onset of symptoms typically occurs in

early childhood; however, genetically confirmed early-adult onset cases have been reported. Since the discovery of the underlying molecular defect in this disorder, high-dose riboflavin supplementation has been reported to be an effective treatment. Familial amyloid polyneuropathy Familial amyloid polyneuropathy FAP , or transthyretin amyloidosis, is characterized by amyloidosis, the buildup of abnormal amyloid protein deposits in the body. FAP can present with progressive axonal sensory autonomic and motor neuropathy and infiltrative cardiomyopathy. Additional features may include conduction block, nephropathy, vitreous opacities, or neurological symptoms related to CNS amyloidosis. Transthyretin amyloidosis can also be an acquired condition, due to age-related deposition of TTR, referred to as senile systemic amyloidosis. Spinal muscular atrophy Spinal muscular atrophy SMA is a neuromuscular disorder caused by the loss of motor neurons within the spinal cord, which results in progressive muscle weakness and atrophy. Other features of SMA may include muscle fasciculations, tremor, poor weight gain, sleeping difficulties, pneumonia, scoliosis, joint contractures, and congenital heart disease. Four clinical SMA subtypes have been distinguished: For each condition, the table below shows the percentage of clinical cases in which a pathogenic variant is expected through analysis of the genes on this panel.

DOWNLOAD PDF PT. 1. PANEL DISCUSSION WITH EDITORS ET AL.

Chapter 4 : DigitalEd Panel: The Value Of Attention: Metrics, Methods and Outcomes

The safety symposium will feature a panel discussion including the editors from the 3 most prominent and respected safety publications in the country: The editors are David Johnson of ISHN, Dave Blanchard of EHS Today and Jerry Laws of Occupational Health & Safety.

We and our trusted partners use cookies and tracking technologies to create custom content for your enjoyment and to provide advertising in line with your interests. CBR â€" Privacy Policy We respect your privacy and we are committed to safeguarding your privacy while online at our site. The following discloses the information gathering and dissemination practices for this Web site. This Privacy Policy was last updated on May 10, Personal Data Collected When you visit our Website, we collect certain information related to your device, such as your IP address, what pages you visit on our Website, whether you were referred to by another website, and at what time you accessed our Website. We do not collect any other type of personal data. Log Files Like most standard Web site servers, we use log files. We and some of our business partners for example, advertisers use cookies on our Website. These cookies track usage of the site for security, analytics and targeted advertising purposes. We use the following type of cookies: Analytics and performance cookies: Depending on your social media setting, the social media network will have record of this and may display your name or identifier in relation to this action. Advertising and targeted advertising cookies: If you wish to disable cookies, you may do so through your individual browser options. For further information regarding cookies and how to manage them, please see http: Pixel tags We use pixel tags, which are small graphic files that allow us and our trusted third party partners to track your Website usage and collect usage data, including the number of pages you visit, the time you spend on each page, what you click on next, and other information about your Website visit. Advertisers We use third-party advertising companies to serve ads when you visit our Web site. These companies may use information not including your name, address, email address or telephone number about your visits to this and other Web sites in order to provide advertisements about goods and services of interest to you. If you would like more information about this practice and to know your choices about not having this information used by these companies, click here. Advertisers, as third-party vendors, use cookies to collect usage and demographic data in order to serve ads on our site. Users may opt out of the use of the DART cookie by visiting the Google ad and content network privacy policy. We use the following advertisers:

DOWNLOAD PDF PT. 1. PANEL DISCUSSION WITH EDITORS ET AL.

Chapter 5 : Pulmonary Rehabilitation

Abstract Panelists: Peter Adams, Physical Review B Irwin Oppenheim, Physical Review E & Massachsetts Institute of Technology Jack Sandweiss, Physical Review Letters & Yale University Reinhardt Schuhmann, Physical Review Letters The panel will include Editors from Physical Review Letters, Physical Review B, and Physical Review E.

The Value Of Attention: Metrics, Methods and Outcomes Moderator: Jason Alcorn, MediaShift Panelists: Measuring and valuing audience attention in your organization. Getting this right allows you to connect with readers the moment it matters. This free online panel is sponsored by Parse. All attendee emails will be shared with the webinar sponsor. Journalists, editors, growth managers, social media editors, marketers, publishers, non-profits, and content creators interested in learning about engagement metrics. Register now for the online panel! Free registration for BigMarker is required. Jason Alcorn is the metrics and impact editor at MediaShift. As a consultant he advises news organizations on business strategy and leadership and works with funders to develop program strategies. Jason lives in Washington, D. You can follow Jason on Twitter at jasonalcorn. You can follow her on Twitter clareondrey. Evan Mackinder is a senior audience engagement editor at Slate. Previously he was the digital engagement manager at the Sunlight Foundation and outreach coordinator at the Center for Responsive Politics. You can follow him on Twitter evandmac. He leads social media strategy, writes The Weekly Reveal newsletter and helps spearhead a variety of audience engagement initiatives. His reporting for Reveal has spanned a variety of topics, including law enforcement, cybersecurity policy and the opioid crisis. You can follow him on Twitter ByardDuncan. We offer real-world training for the digital age.

Chapter 6 : Doomsday Clock #7, Annotated, Part 1 â€“ The Multiverse, The Legion & The Jokermobile

Northwest Editors Guild Navigation. About the Guild. Meetings & Events; Nonfiction Publishers: A Panel Discussion. This content is only available to current members.

Chapter 7 : Homepage | College of American Pathologists

The panel discussion looked at three real-world examples of MCDA application and how MCDA can be applied and what lessons beta-testing has taught us: The first example examines the application of MCDA to health technology assessment (HTA).

Chapter 8 : Test | Invitae Comprehensive Neuropathies Panel

A candid panel discussion on ET ALIEN disclosure with prominent UFO researchers: Richard Dolan, Stanton Friedman, Linda Moulton Howe, Grant Cameron and Victor Viggiani.

Chapter 9 : "Library Discussion Panel Part I" by Lowell Walters, Carl Merat et al.

This panel discussion will feature experienced local editors sharing their insights on the freelance editor www.nxgvision.com will discuss topics such as finding clients, setting rates and business matters such as marketing.