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Chapter 1 : AP Cardiology â€“ Cardiology podcast for internists, residents and medical students

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Print Version Transcatheter placement of an intracoronary stent s , percutaneous, with or without other therapeutic intervention, any method; single vessel Transcatheter placement of an intracoronary stent s , percutaneous, with or without other therapeutic intervention, any method; each additional vessels Percutaneous transluminal coronary balloon angioplasty, single vessel Percutaneous transluminal coronary balloon angioplasty, each additional vessel Percutaneous transluminal coronary atherectomy, with or without balloon angioplasty, initial vessel Percutaneous transluminal coronary atherectomy, each additional vessel Correct reporting of interventional cardiology services requires knowledge of cardiac anatomy as well as an understanding of CPT coding conventions. Only the most highly valued procedure stent code in above listed codes would be reported with the initial vessel code in the first vessel. Any other therapeutic coronary artery procedures ex, angioplasty or athrectomy in different vessels are reported using the "each additional vessel" code for the same procedure, which is reimbursed significantly less than the initial vessel code. If a single intervention is utilized in more than one of these three vessels, the first vessel is to be identified using the respective "single vessel" code. Each additional major coronary artery treated is identified by using the "each additional vessel" code. Interventions in the branch vessels are considered a part of the intervention in the major vessel and are not reported separately. Anatomic variants should be reported as closely as possible to a corresponding major vessel and not separately coded. The Medicare Correct Coding Initiative CCI defines a hierarchical schema in technical complexity that exists when multiple coronary interventions are performed in a single session. This means that certain services supersede other services and the other services are not reported separately. Generally, stent placement supersedes atherectomy, which supersedes angioplasty. Many payors, including Medicare, recognize only three major coronary arteries. The left main coronary artery is not recognized as a separate major coronary artery. The description below shows the three major coronary arteries recognized by Medicare. RC Right Coronary includes: Diagonal Branch 1 and Diagonal Branch 2, and Septal branches If the physician places a stent in the left circumflex and performs an angioplasty in the obtuse marginal 1 which is a branch of the circumflex , only the stenting should be reported LC. Stenting of the right coronary artery and left circumflex coronary artery. Note that if multiple stents were placed side by side in a single vessel, the stent code would be reported only once. If a diagnostic cardiac catheterization was performed prior to intervention, it is coded secondary with the appropriate catheterization code and modifiers and Only one of the three most commonly performed therapeutic coronary techniques stenting, atherectomy, and angioplasty can be reported in each major coronary artery and its branches , even if more than one technique is performed. Stenting includes any angioplasty or atherectomy that is performed in the same coronary artery, and atherectomy includes any angioplasty that is performed in the same coronary artery. Codes and , designated as add-on codes, should be reported in addition to the primary procedure eg, angiography, angioplasty without appending modifier Intravascular distal coronary blood flow velocity is performed during a cardiac catheterization procedure for coronary angiography or other therapeutic intervention. Assign modifier for physician billing. Code is reported for atherectomy of a single coronary artery. Like other add-on codes, code is exempt from modifier 51 Multiple procedures. Both of these codes are assigned per vessel; therefore, if multiple lesions are treated in the same vessel or its branches , only one atherectomy is coded. There are different types of athrectomy catheters, each is a different method for removing plaque. Directional where a cup-shaped blade shaves plaque off the vessel ex, Flexi-cut catheter Extraction uses blades to shave plaque off vessels and then vacuums out ex, TEC catheter Rotational device uses a rotating bur to abrade plaque from vessel wall and suction the debris out ex, Rotablator Laser athrectomy uses a laser-tipped catheter to ablate the plaque ex, Vitesse excimer laser system Examples: Intra-Aortic Balloon Pump is a device used to improve cardiovascular function. Medical Necessity It has never been more important for health care organizations to be proactive with respect to assurance of medical necessity. Department of Health and Human Services Office of Inspector General HHS OIG , the most

common Medicare reimbursement violation is the failure to comply with the medical necessity requirements, especially for certain costly diagnoses involving interventional cardiology procedures for example, angioplasty and pacemaker implantation. Document what was done and medically necessary and assign codes to reflect the medical record documentation. If you have questions or comments about this article please contact us. Comments that provide additional related information may be added here by our Editors. Medicare may have restrictions on the use of a modifier used to bypass an edit. This important part of coding can alert a payer or Each code is listed with the following information: Sometimes that places us in the role where we need to save our clinicians from themselves, and the patterns they have fallen into We have been receiving several DDS referrals to our massage therapists who do intra-oral work. I am a certified DOT medical examiner and have applied to get my CLIA lab waiver for urinalysis, finger prick blood tests for A1c, cholesterol and glucose. I realize I cannot diagnose patients with these tests, but I am using them to make decisions in the DOT process and with

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Chapter 2 : Cardiology Section II - Part 2 - Physeo

An acute electrolyte abnormality would most likely be suspected if the ECG shows: A) the presence of U waves or peaked T waves. B) the presence of J waves or ST segment elevation.

Last month we chatted with Dr. Stephen Sinatra about new nutrients to keep the heart and arteries healthy, particularly a nutrient called propionyl-L-carnitine PLC. This month, we will focus on metabolic cardiology and how adenosine triphosphate ATP , and thus its precursors, the carnitines, CoQ and D-ribose, is the bottom line in heart health. Anybody with any type of cardiovascular disease has a heart that is always leaking ATP. It takes too long and the ATP leakage is faster. Cardiologists are always thinking about oxygen in the heartâ€"and oxygen is importantâ€"but oxygen is only the stepping-stone to ATP. Oxygen is only a toolâ€"but a vital oneâ€"in the production of energy via ATP. The chemicals would just sit there, unreacted. The energy needed to cause the chemicals to react is obtained from the release of chemically stored energy. Energy is a force, not a compound. In previous chats with Dr. Sinatra, we have discussed the emerging field of metabolic cardiology and how it is saving lives and improving the quality of life in heart patients. Metabolic cardiology is a sub-specialty of cardiology that deals with the very core of heart disease itselfâ€"the biochemical changes in cardiac cells by which energy is provided for vital processes and activities. Actually, metabolic cardiology is more than energizing and powering the heart because the same factors energize and power all of the cells in the body. Metabolic cardiology applies to preventing and overcoming fibromyalgia, chronic fatigue and Syndrome X, just as it does to all aspects of heart disease. In the introduction to Dr. Jim Roberts points out several shortcomings of conventional cardiology and how it was missing the boat in terms of not being able to stop the continual decline in heart function even after arteries had been stented or by-passed and proper medications given. Why was there a functional delay in recovery? The ischemia â€¦ burns off the adenine nucleotide pool, the source of cellular energy. He also is certified in anti-aging medicine. He is a fellow of the American College of Cardiology and former chief of cardiology at Manchester Memorial Hospital where he was director of medical education for 18 years. Sinatra is also assistant clinical professor of medicine at the University of Connecticut School of Medicine. Sinatra integrates conventional medical treatments for heart disease with complementary nutritional, anti-aging and psychological therapies that help heal the heart. He is uniquely qualified to give advice on nutritional supplements and the heart. Sinatra is one of the few medical doctors who formulates his own vitamins. He is expert in dosage, absorption, how to pick quality ingredients, and the effects of combining supplements with cardiac medications. His most recent book is the aforementioned The Sinatra Solution. Additional information can be found on his website at www. You are always at the forefront of metabolic cardiology. How did the evolution of your research and therapies come about? I have been using CoQ for some 20 yearsâ€"ever since around the time that Dr. Emile Bliznakov came out with his book The Miracle Nutrient: I have been using CoQ in my practice since or I have been using the carnitines for 10 years. Specifically, I used L-carnitine at first, then added acetyl-L-carnitine about five years ago. I have been aware of the role of L-carnitine in the mitochondrial turnover of ATP since the late s or early s. There have been many very interesting studies published on the carnitines in the cardiovascular journals since then about how heart cells deficient in carnitine and CoQ cannot perform adequately. Now an improved heart-specific carnitine called propionyl-L-carnitine PLC is available and my patients are gaining even more benefit. I have been using D-ribose for about three years now. I learned about D-ribose at a meeting on aging from fellow cardiologist Dr. Jim Roberts, who wrote the introduction to my book. Coenzyme Q, collectively carnitines as there are three carnitines. There is L-carnitine, acetyl-L-carnitine which gets more into the brain than the heart, and there is PLC, which has a very fast half-life the time required for the quantity to fall to half of its initial value. PLC gets inside the heart and other muscles both peripheral muscle and cardiac muscle â€"this is a very heart-selective carnitine. The third nutrient in the Triad is D-ribose, which really provides the precursor. The same thing is true of L-carnitine, and the same thing also is true of D-ribose. All of these studies have been done individually where you can do a crossover study, double-blind, do a washout, and all of a sudden you get a minute longer on the treadmill by using one of these nutrients. No one, to my knowledge

has done a study of all three nutrients together. I want to do such a study to see if the combination synergistically results in even a longer time. They are going to give people a much better quality of life and reduce a lot of human suffering. Now, if you do a biopsy on ailing hearts, and you measure carnitine and CoQ levels, they are diminished. That literature is old. Folkers showed this about 40 years ago. So I have been using magnesium for some 30 years. Most doctors are already familiar with magnesium. As for my evolving awareness of these nutrients, carnitine and CoQ were literally placed in my path. I read the literature and, as a cardiologist, I wanted to see if they would help my patients. As you know from writing your books, the best way to become very informed on a subject is to give lectures to other cardiologists wishing to know more about your treatment and then write a book on that subject. Publishers asked me to gather my lectures in a more reader-friendly format for both patients and cardiologists. Writing the books made me really appreciate the complexities of the nutrients. One thing I did learn from writing the books and using the therapies on thousands of my patients and subscribers to my newsletter, many of whom write to tell me about their success with carnitine and CoQ and the positive feedback about what these substances have done for their lives. So when you take in the literature, and use the nutrients every day in your practice, you get a flavor of how these vital nutrients are really working. You mentioned that there is quite a bit about CoQ and L-carnitine in the medical literature. Yet, if I were to pick up the phone book and call every cardiologist in my area, I doubt that I would find more than one in who knew anything about L-carnitine and heart disease. Nutrients have no such patent protection and resulting profit. Yes, this is how many, if not most, doctors receive their continuing education in this country. But this is not going to happen with nutrients because of the patent issue that limits the profitability that supports this type of education. Meanwhile, there is a glimmer of hope. The American College of Nutrition has an exam for any doctor who wants to become certified as a certified nutrition specialist CNS. They can take this exam. I can tell you this: I am on this examination committee, and I will lobby to have these nutraceutical substances on the exam. I think they are so vital for cardiovascular and general health that they need to be included on board examinations so that physicians come to understand their vital interplay. You are also invited to speak at various meetings and symposia of physicians who are also very interested in complementing their treatments with nutrients. Thus, you are constantly educating physicians about how to complement their practice with nutrients. Still, you, personally, can reach only a minority of those who should be learning about the benefits of these heart-healthy, artery-healthy nutrients. Your books certainly help to educate a few physicians who have open minds, but I believe that, possibly even more important, are the testimonies of your readers and patients who call their doctors to explain how CoQ, or carnitine, or D-ribose, or fish oil have helped them. In addition to your books and newsletters, you have been kind enough to discuss these topics with us over the years. I have posted many of our chats on my website at www. I particularly would like to refer our readers to the following topics: When I lecture at these conferencesâ€”and the conferences are being attended by more and more doctorsâ€”there are now about 3,, doctors who go to these conferences. In fact one cardiologist told me the other day that one of his patients gave him my book. Like the rest of us, doctors tend to respect people who are just like they are, and who have had the same kind of training and initiation. I think the pendulum is starting to turn. Instead of, as you said, finding one in who knows about nutritional therapies, now it might be five or 10 out of I have been taking care of cardiac patients for 30 years, and I am only now beginning to realize what the bottom line might be. It was almost as if a light bulb was switched on over my head! Here, then, is what I believe the bottom line of metabolic cardiology can be: It takes too long and the ATP leakage is faster than the replacement process. These patients cannot get enough ATP in their cardiac cells because the ischemic or compromised heart is always leaking ATP that it is gone from the cytoplasm forever! It is burned to uric acid and other metabolites. What I have learned as a clinical cardiologist is that it is all about ATP. Perhaps I should repeat my theme on ATP once again. What happens in ischemia is that the Krebs cycle citric acid cycle shuts down. Yes, basically, the Krebs cycle becomes poisoned, so to speak. Then AMP is formed. With the high levels of AMP, this diffuses leaks out into the cytoplasm of the cell. The last-ditch effort of the cell in ischemia is to form adenosine. Adenosine is like a vasodilator to keep the cell fortified with oxygen.

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Chapter 3 : Treatment of Acute Heart Failure

Drs. Deepak Bhatt and Peter Block discuss IVUS guidance of coronary DES implantation in an all-comers patient population (ULTIMATE); and high-speed rotational atherectomy prior to DES implantation in severely calcified coronary lesions.

List at least four common symptoms of ischemic heart disease. Name three types of vasodilator drugs. List at least three imaging modalities used to diagnose aortic stenosis. Name four non-pharmacologic therapies used to treat systemic hypertension. Describe three cardiac arrhythmias that precipitate congestive heart failure. Name the two basic components of an implantable cardioverter defibrillator. Name two imaging modalities used to diagnose pulmonary hypertension. List at least six risk factors for bleeding when using anticoagulants. Name at least five types of benign cardiac tumors. List four normal diagnostic test findings in pregnancy using electrocardiogram. This test is a multiple choice, open book test and contains questions. This is a hard cover book and has 36 chapters and pages. This course is Part Two of a larger book. In this part you are only tested on the last half of the book. When you order a complete course, you will receive a text book, question booklet and answer sheet. Your order will ship within 24 hours of receipt of order and you should receive your order by mail in business days. When you order a test only for mailing, you will receive a test booklet and an answer sheet. Your order will ship within 24 hours of receipt of order and you should receive your order by Fedex within the timeframe for the shipping option you chose. Please note that if you order the test only option you will NOT receive a course book. When you order an Ebook, you will log in to your account profile and download the Ebook, test booklet and answer sheet. For further instruction, please view our instructional video. If you would like to choose the download test option, you will log in to your account profile and download the test booklet and answer sheet. For further information, please view our instructional video. If you plan to take the test online, please be sure you have the course book, as the online testing option does not include the course book. If you would like to have this test faxed to you, please be sure that you have the textbook and question booklet. You will receive your test the same day. If you have any questions, please call our customer service department at

Chapter 4 : AIR Series: Cardiology - CHF, PVD, and Others (part 2)

CPT Cardiovascular Surgery Part 2 with guideline breakdown.

Pulsus alternans Impaired end organ function, such as decreased urine output There are no physical exam findings that individually establish a definitive diagnosis of AHF, but relevant findings may be considered in two main categories. First, there are findings that suggest the presence of underlying cardiac dysfunction that provides the substrate for the AHF episode. Second, physical exam signs can support the diagnosis of AHF by suggesting the predominant pathophysiologic process involved in the decompensation, such as volume overload peripheral edema, pleural effusions, rales, ascites, elevated jugular venous pressure, hepatojugular reflux , vascular redistribution elevated jugular venous pressure, hepatojugular reflux, often elevated blood pressure , or low cardiac output cool extremities, low blood pressure, often narrow pulse pressure, pulsus alternans. Note that patients with chronic heart failure may have markedly elevated pulmonary venous pressure causing significant dyspnea with relatively mild rales. Patients with AHF due to vascular redistribution frequently have rapid onset of symptoms that can occur in the absence of signs of marked volume overload. What diagnostic tests should be performed? What laboratory studies if any should be ordered to help establish the diagnosis? How should the results be interpreted? As with physical exam findings, laboratory tests can provide insight into the diagnosis of AHF. Plasma concentrations of B-type natriuretic peptide BNP and its by-product, N-terminal-proBNP NT-proBNP , are increased in the presence of elevated ventricular both left and right pressure and volume, and have been demonstrated to assist in the diagnosis of AHF in patients with dyspnea. Note specifically that elevated BNP levels can occur with right-sided heart failure, and may be present in acute pulmonary embolus. Hyponatremia may be suggestive of neurohormonal imbalance due to advanced or undertreated chronic heart failure or iatrogenic from diuretics particularly thiazides. Hyperkalemia often suggests acute renal insufficiency due to worsening heart failure, but may also be due to recent initiation of a mineralocorticoid receptor antagonist spironolactone or eplerenone , ACE inhibitor, or angiotensin receptor blocker while hypokalemia is frequently seen in the presence of increased diuretics with insufficient potassium repletion. Hypomagnesemia may accompany hypokalemia, especially if diuretic-related, and should be assessed. Hyperchloremia may be associated with excessive diuresis and volume depletion. Blood urea nitrogen BUN and creatinine serve as markers of renal function. Chronic renal insufficiency is a frequent comorbidity in patients with heart failure and AHF episodes are often accompanied by acute on chronic renal failure. Cystatin-C is another marker of renal function with the putative advantage of responding more rapidly to acute changes in renal function. Other markers to assess acute kidney injury are in development and should assist the evaluation and treatment of these patients. What imaging studies if any should be ordered to help establish the diagnosis? The ECG can assist in the evaluation of myocardial ischemia, left ventricular hypertrophy, arrhythmias, and electrolyte disorders as contributing factors to AHF. The chest x-ray CXR is useful in assessing the presence and extent of pulmonary congestion, although it may underestimate the extent of congestion and pulmonary venous hypertension in patients with chronic heart failure. Pleural effusions may be detected, as well as evidence of pneumonia, a common confounding differential diagnosis, comorbidity, or exacerbating factor. Changes in cardiac silhouette, suggestive of pericardial effusion, or aortic contour e. In patients with no prior echocardiogram, this test is probably the most useful noninvasive imaging study, given that it provides information on size, structure, and function of all cardiac chambers and valves, potential wall motion abnormalities, and estimates of hemodynamics, including central venous and left ventricular filling pressure. However, most patients do not require an emergent or urgent echo to guide early therapy, and a full, more detailed, higher quality, and clinically relevant echocardiographic examination can be performed after compensation is reestablished. One study demonstrated no significant difference in left ventricular ejection fraction upon presentation with AHF compared to later in the hospitalization. The PA catheter is an essential and very useful diagnostic tool in properly selected patients. In patients with evidence of shock in whom it is unclear whether the etiology is cardiogenic or noncardiogenic, invasive assessment of central venous pressure, pulmonary arterial PAP and

pulmonary capillary wedge pressures PCWP , cardiac output, and vascular resistances can provide crucial information to guide appropriate selection of and evaluate response to therapy. A PA catheter should not be routinely used in most patients with AHF, given the small, but finite, risk of infection, vascular injury, and other associated complications. One of the greatest problems with the use of the PA catheter is that members of the medical team may lack familiarity with wave forms and have limited experience in distinguishing artefact and recognizing other problems e. Vigilance in assessing data obtained from the PA catheter and interpretation in the context of other data from the patient is essential to ensure that information used to guide clinical decisions is correct. Other diagnostic tests, such as noninvasive cardiac stress testing or cardiac catheterization with coronary angiography, should also be considered in selected patients. The first step in management of the patient with AHF is to address life-threatening issues, including, but not limited to: The most common presenting symptom of subjects with AHF is dyspnea and respiratory failure is the most frequent life-threatening condition for these patients. Immediate administration of the following is recommended: If it is safe to do so, support the patient in assuming an upright, sitting posture. Many patients will do this on their own to optimize their ventilator efficiency Oxygen: Although no randomized study has been performed, immediate administration of supplemental oxygen is the most readily available means to increase end organ oxygen delivery. If the above measures remain inadequate, rapid application of noninvasive ventilatory support CPAP or NIPPV has been shown to be very effective in rapidly improving symptoms, hemodynamics, and metabolic abnormalities associated with AHF. If noninvasive measures are insufficient, rapid intubation with mechanical positive pressure ventilation should be employed. Sublingual or intravenous nitrates can be very effective as vasodilators, decreasing pulmonary venous pressure and relieving dyspnea. Rapid administration of intravenous nitrates in patients with severe pulmonary edema decreased the need for mechanical ventilation and myocardial infarction compared to a high-dose furosemide strategy in a randomized study. Most patients will also have significant volume overload contributing to the respiratory insufficiency, so if there is evidence of volume overload as opposed to volume redistribution , rapid administration of intravenous loop diuretics is recommended. Although it has remained controversial, one early study suggested that furosemide also directly dilates pulmonary veins. Opiates morphine in the setting of AHF have been associated with increased rates of mechanical intubation, prolonged hospitalization, more frequent ICU admissions, and higher mortality. While this association may be reflective of the greater disease severity of patients receiving morphine, these findings argue against routine use of morphine in patients with dyspnea, as well as for careful monitoring in the select patients who receive opiates. All of these agents increase cAMP and intracellular calcium, with the related increases in heart rate, myocardial oxygen consumption, and arrhythmias, potentially resulting in myocardial ischemia, infarction, or death. Monitoring of response to these therapies depends upon the initial derangements, including blood pressure, peripheral perfusion, respiratory status, urine output, mental status, and other end organ function. Catecholamines such as dobutamine, epinephrine, and norepinephrine have been used to improve myocardial contractility and increase the heart rate in the setting of AHF. Dobutamine is a predominant beta-1adrenergic receptor agonist with mild vasodilating properties. Low doses of dobutamine are also associated with mild vasodilation, while higher doses can cause vasoconstriction. Phosphodiesterasetype III inhibitors, such as milrinone in U. Many clinicians no longer use a bolus loading dose of these agents, so as to avoid significant hypotension, but a bolus may be used in selected situations. Hypotension, tachycardia, atrial, and ventricular arrhythmias, increased myocardial ischemia, and other adverse effects, including suggestions of increased mortality in patients with ischemic heart disease, may occur and patients should be carefully monitored. Levosimendan available in Europe and other non-US countries is an ATP-dependent potassium channel activator with myocardial calcium sensitizing effects and possible PDE III inhibitor effects , and acts as a vasodilator and inotrope. Many clinicians no longer use a bolus loading dose, so as to avoid significant hypotension, but a bolus may be used in selected situations. Hypotension, tachycardia, atrial and ventricular arrhythmias, and other adverse effects may occur, and patients should be carefully monitored. Intraaortic balloon pumps IABP can be used at many centers in the setting of severe cardiac compromise and provide a rapid decrease in ventricular afterload with some augmentation of forward flow. Other mechanical support devices may be used

at specialized centers, including ventricular assist devices VADs and extracorporeal membrane oxygenation ECMO. Fulminant renal failure requiring emergent treatment is uncommon in patients with AHF, but significant renal insufficiency is quite frequent. Emergent hemodialysis may be used to treat life-threatening electrolyte abnormalities or other severe sequelae of renal failure. Ultrafiltration has been increasingly used to treat patients with significant volume overload. Some centers will initiate veno-venous ultrafiltration in patients with marked fluid overload i. Clinical trials are currently underway to provide evidence regarding these strategies. Careful attention should be given to the filtration rate so as to not exceed the vascular refill rate. Some clinicians will monitor for hemoconcentration, presumably to avoid excessive or too rapid volume removal. Nonetheless, many clinicians do not routinely use dopamine in the setting of mild-to-moderate renal insufficiency, and most reserve it for patients who fail diuretics. This strategy is also being evaluated in ongoing clinical studies. Diuretics remain the most commonly administered agent for AHF. Intravenous furosemide or equivalent can provide rapid symptom relief as well as decrease the underlying volume overload. Diuresis can also frequently improve renal insufficiency, since the most common cause of acute renal insufficiency in AHF patients is increased central venous pressure. Patients receiving high-dose diuretics appear to have more rapid symptom relief with mild transient worsening of renal function, and there does not appear to be a difference between continuous infusion versus bolus-dosing strategies in a recent clinical study. These findings suggest that the strategy that is most appropriate to ensure reliable diuresis at the specific clinical setting should be implemented. While most patients present with volume overload, there is agroup of patients with AHF due to volume redistribution, and these patients will generally not benefit from aggressive diuresis. Electrolyte repletion is essential in patients with hypokalemia and hypomagnesemia. Many patients with AHF will present with a history of multiple concomitant medications, including therapies for chronic heart failure. Depending on the severity of the AHF episode, most of these medications can and should be continued during their hospitalization. Beta-blocker therapy in particular should be continued during the hospital course, unless there is cardiogenic shock, symptomatic bradycardia, or advanced heart block. Patients in whom beta-blocker therapy is discontinued during hospitalization have worse clinical outcomes, even when adjusted for disease severity. ACE inhibitors and angiotensin blockers may be held in the context of acute renal failure, but should be resumed as soon as possible. Physical Examination Tips to Guide Management. The physical exam may be used to assess the response to therapy and guide management. Blood pressure should be carefully followed, though with the advent of improved noninvasive monitoring, invasive arterial lines are rarely necessary. Hypotension has been associated with poor outcomes in the setting of AHF, and iatrogenic hypotension should be assiduously avoided. Hypertension can be one of the major precipitants of AHF and should be treated. Heart rate is often a reflection rather than a cause of the AHF episode, and the initial tachycardia often improves in conjunction with the improvement in dyspnea. However, atrial fibrillation with rapid ventricular response is a well-known precipitant of AHF. Tachycardia may occur due to the positive chronotropic effects of some drugs e. Bradycardia is less common and may be due to excess beta-blocker therapy. Respiratory rate is often not as carefully assessed clinically, and may not be as reliably sensitive to therapy as other vital signs. Tachypnea may represent inadequate resolution of the initial episode of dyspnea or a new event, such as a pulmonary embolus. Fever is suggestive of underlying infectious process, particularly pneumonia or urinary tract infections, both of which can instigate AHF exacerbations. Oxygen saturation may be measured noninvasively and is useful to follow in patients with marginal oxygenation. Body weight is frequently overlooked in the assessment of the patient until later in their hospital course, yet can be a very useful measure to follow the response to therapy. Body weight should be obtained as early as practical in the hospital course. Urine output and daily input and output measures are also useful corroborative measures, but in many clinical practices appear to be less reliable. Jugular venous pressure JVP is by far the most useful, and most challenging, physical examination finding to monitor response to therapy in the AHF patient. Elevation of the JVP suggests that there is persistent volume overload and additional diuresis is indicated, whereas a low JVP suggests that there may be excessive volume depletion.

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Chapter 5 : FMC Acquires Turner White Communications - Frontline Medical Communications

Cardiology Part 2 - AIR STAMP OF APPROVAL AND HONORABLE MENTIONS Below we have listed our selection of the 12 highest quality blog posts within the past 12 months (as assessed in August) related to toxicology topics, curated and approved for residency training by the AIR Series Board.

The Unhealthy Heart By age 36 my life was a wreck because my heart was in shambles. Prideful, arrogant, unloving, selfish, cruel, deceptive, and separated from God who created it. Everything I was putting into my heart was unhealthy and as a result so was just about everything coming out of it. I was internally miserable and a very uncaring and selfish person even if things appeared from the outside to be good in my life. I loved and hated myself at the same time. My heart was a bomb ready to explode. My loving heavenly Father and Lord Jesus Christ, whom I mocked and cursed with foul language on a regular basis, were about to save me by their grace and mercy. If the spiritual heart is the real person that we are before God and the source and control center of our lives, then we certainly want it to be healthy! This week the example of an unhealthy heart will be from an unsaved non-Christian. I will be the example for both. I was focused on myself and the things of this world money, prestige, possessions, pleasure, vacations, etc. I had rotten fruit lawlessness while they had plastic and pretend fruit that looked real but was really imitation legalism. Both my religious friends and I were selfish in our motives. True agape love unselfish giving , which only comes from God, was absent because God was absent from my life in a real personal relationship. I had anger issues originating from fear. Fear of death, fear of losing all my idols money, prestige, being young and healthy, etc. Nothing could quench my inner thirst which led to frustration and depression. My heart was spiritually dead and separated from God. It was incurable and diseased far beyond any treatment that modern medical science could provide. For 36 years my entire culture and personal experiences did not teach me about a real and personal God who wanted to be the center of my life. It had been etched on my heart that God was either unknowable, non-existent, or irrelevant to daily life. The diagram below summarizes the state of an unhealthy non-Christian heart. It is designed for teaching purposes. No single individual would likely have all of these characteristics, but I had most of them. It shows behavior of religious and non-religious people.

Chapter 6 : ESC/EACTS Guidelines on Myocardial Revascularization - Part

Which of the following waves/elevations are seen on ECG readings in patients with non-ST-segment elevation myocardial infarction (NSTEMI)?

Chapter 7 : Part Two: Metabolic Cardiologyâ€"Itâ€™s All About ATP

The Mounted CP Stentâ„¢ consists of a bare CP Stentâ„¢ pre-mounted on a BIB Catheter. This system allows the physician the flexibility of using the pre-mounted complete system and will save the time required to mount the stent on the catheter.

Chapter 8 : ESC/ESH Guidelines on Arterial Hypertension - Part

The following are summary points from part 2 of a 2-part series on the spectrum of restrictive and infiltrative cardiomyopathies: Endomyocardial diseases are another rare cause of restrictive cardiomyopathy (RCM); the most common is endomyocardial fibrosis (EMF).

Chapter 9 : Cardiology Diagnosis & Treatment P2 Category A credits

Part 2 of this course covers all the major disease entities and therapeutic challenges in cardiology. Concise, yet comprehensive, it is perfect for all aspects of heart disease care.