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Target audience	This activity is designed to meet the educational needs of physicians, PAs, nurses, nurse practitioners and other HCPs who have an interest in EBM.
Learning objectives	At the end of this educational activity, participants should be able to: • Explore the educational content surrounding the use of IV iodinated contrast in chronic kidney disorder (CKD) from radiology and nephrology. • Review pharmaceutical recommendations for the management of perioperative gabapentinoids linked with respiratory complications and the effectiveness of behavioral therapy for men with symptoms of overactive bladder. • Apply medical management principles grounded in evidence-based medicine regarding CPAP versus standard of care in mild obstructive sleep apnea and review of echocardiogram in the diagnosis of syncope in patients with normal heart exams and ECG.

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Use of IV-iodinated contrast in CKD — New joint consensus statement from radiology and nephrology[1]

Use of IV contrast is an important area where clinical practice has lagged behind the evidence and has created barriers to optimal patient care. Historical data suggesting that IV contrast caused acute kidney injury (AKI) was confounded as these studies were usually done in ill patients seen in the ER or the hospital with many other potential causes for AKI being present. High quality recent data has dispelled the fear that IV contrast poses a significant risk for AKI but latent bias has persisted and prevented new practice algorithms from being deployed. Earlier this year, the American College of Radiology and the National Kidney Foundation released a joint statement on the use of IV contrast in patients with kidney disease and serves as the basis for this review. All studies referenced below can be found in the joint statement.

To clarify the above situations where acute illness, dehydration, use of nephrotoxic drugs, etc. are the likely cause of AKI around the time of IV contrast administration, this clinical picture has been labelled **contrast associated** AKI (CA-AKI). This is to specifically highlight that in these situations, the contrast is not felt to be contributory to the AKI. On the other hand, the rare circumstances where IV contrast, if felt to be the etiology, are termed **contrast induced** AKI (CI-AKI). The consensus statement asked a series of relevant questions and followed with evidence-based answers. Throughout the statement, CA-AKI is differentiated from CI-AKI since the kidney injury in the former is not felt to be related to IV contrast. The most important questions are as follows:

What Is the risk of CA-AKI and CI-AKI in patients who have stage 1 through 4 CKD?

- The risk of CA-AKI ranges from 5% at an eGFR >60 mL/min up to 30% for an eGFR <30mL/min. This risk is much higher than the risk of CI-AKI because it includes any and all causes of AKI coincident to contrast media administration, even though the contrast is not felt to be etiologic to the AKI.
- The risk of CI-AKI is substantially less than that of CA-AKI. However, the actual risk has not been consistently quantified in patients with severe pre-existing kidney disease. Importantly, several large controlled observational studies have shown no evidence of CI-AKI regardless of CKD stage, whereas others found evidence of CI-AKI only in patients with severely reduced kidney function. In such studies, the risk of CI-AKI has been estimated to be near 0% at eGFR greater than or equal to 45, 0%–2% at eGFR of 30–44, and 0%–17% at eGFR <30 mL/min.

What other major patient-related factors increase the risk of CA-AKI or CI-AKI?

- **CA-AKI.** Multiple patient-related risk factors have been associated with CA-AKI. The primary risk factor is a baseline reduced eGFR, with some studies finding an additive risk of CA-AKI from diabetes mellitus. Additional risk factors include nephrotoxic agents and exposures, hypotension, hypovolemia, albuminuria, and impaired kidney perfusion (e.g., congestive heart failure.) Although multiple myeloma has long been considered a risk factor for CA-AKI, this is not supported by more recent literature.
- **CI-AKI.** Few studies have linked patient-related risk factors with CI-AKI. In studies that did find evidence of CI-AKI, the primary risk factor was a baseline, reduced eGFR. No other factors that increase CI-AKI risk beyond eGFR alone have been confirmed in well-controlled studies of intravenous media.

Are there clinically relevant differences in CA-AKI and CI-AKI risk for patients with reduced kidney function with intravenous iodinated low-osmolality contrast media compared with intravenous iodinated iso-osmolality contrast media?

The simple answer for both categories is that there are no relevant differences in risk related to the osmolality of the contrast agent.

Which patients should undergo IV saline prophylaxis to prevent AKI prior to intravenous iodinated contrast media administration?

Prophylaxis is indicated for patients who have had a recent history AKI or a baseline eGFR less than 30 mL/min. However, the evidence supporting this statement is based on data for the general prevention of CA-AKI rather than CI-AKI specifically. Prophylaxis is not indicated for the general population of patients with stable eGFR greater than or equal to 30 mL/min. This eGFR threshold should not be adjusted solely based on concomitant diabetes mellitus. In an observational study of 1,112 patients with stable eGFR of 30–44 mL/min, diabetes mellitus did not independently increase risk of CI-AKI in patients undergoing contrast-enhanced CT. When prophylaxis is indicated, isotonic volume expansion with normal saline is the preferred method.

Should serum creatinine/eGFR screening be used to identify patients at risk for CI-AKI prior to IV contrast?

Routine screening of renal function is not recommended in the consensus statement. Rather, the consensus statement recommends screening based on eGFR to be used to identify patients who may be at increased risk of CI-AKI. A personal history of kidney disease (e.g., CKD, remote AKI, kidney surgery, kidney ablation, albuminuria) is the most useful clinical issue to suggest the need for kidney function measurement. It seems prudent to verify renal function with eGFR within the prior 30 days of test ordering for these patients. Diabetes

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mellitus is an optional factor for screening, although not supported by current data. Patient age and the presence of hypertension, both treated and untreated, are of uncertain utility as independent triggers for kidney function assessment during radiology point of care. They are sensitive indicators and confer a large false-positive rate to the identification of patients with eGFR <30 mL/min. Patients who do have an eGFR <30 mL/min should prompt consideration by the referring provider and radiologist to discuss the risks and benefits of contrast media administration.

Should intravenous iodinated contrast media be withheld in patients with CKD Stages 4 and 5 not undergoing hemodialysis?

Patients with CKD Stages 4 or 5 (eGFRs of 15–29 mL/min) who are not undergoing maintenance hemodialysis are at potential risk of CI-AKI. The number needed to harm from contrast media administration has been calculated in well-controlled observational studies to be as low as six and as high as infinity (i.e., no harm). If contrast media administration is required for a life-threatening diagnosis, then it should not be withheld based on kidney function.

Should any of the above recommendations be altered in patients receiving certain nephrotoxic medications or undergoing chemotherapy, especially if they have normal kidney function?

In general, the above recommendations should not be altered in patients receiving nephrotoxic medications or undergoing chemotherapy. This is especially true for patients who have normal eGFR or mild-to-moderate reductions in eGFR because they are not considered at risk, regardless of the drug(s) prescribed, and therefore do not need eGFR screening prior to contrast administration. However, monitoring eGFR in patients receiving nephrotoxic medications (e.g., aminoglycosides) or undergoing chemotherapy is important before, during, and after treatment to identify incident nephrotoxicity (CA-AKI).

Is there a role for withholding certain medications prior to intravenous iodinated contrast media administration to decrease the risk of kidney injury?

Metformin does not increase the risk of CA-AKI or CI-AKI. Metformin should only be withheld in patients with eGFR <30 mL/min. This is already an FDA guideline for metformin use and therefore not relevant assuming metformin is used in the appropriate patient population with an eGFR >30 mL/min. Also, in patients with an eGFR>30 mL/min, it is not necessary to withhold nonessential potentially nephrotoxic medications (e.g., nonsteroidal anti-inflammatory drugs, diuretics, aminoglycosides, amphotericin, platins, zoledronate, methotrexate). Whether to withhold renin-angiotensin-aldosterone system inhibitors (RAASi) is controversial. A meta-analysis of 12 studies and 4,493 patients found no difference in risk of CA-AKI between patients receiving and patients not receiving RAASi. On the other hand, given the lack of strong evidence demonstrating that continuing RAASi is beneficial, one option would be to withhold RAASi in patients at risk for CA-AKI for at least 48 hours before elective contrast-enhanced CT to avoid the potential for hypotension and hyperkalemia should CA-AKI develop.

In summary

At many practices nationwide it is still a standard of care to avoid IV contrast in patients with an eGFR between 30–60 mL/min. Additionally, many radiologists still request recent renal function monitoring in the absence of an indication, despite this new consensus statement. It is time to advance our clinical practice to match contemporary evidence-based guidelines. The risk of administering modern intravenous iodinated contrast media in patients with reduced kidney function has been overstated. This is primarily because of the conflation of contrast-associated acute kidney injury (CA-AKI) with contrast-induced acute kidney injury (CI-AKI) in uncontrolled studies. In certain high-risk circumstances, IV saline prophylaxis may be considered in patients with an eGFR of 30–44 mL/min at the discretion of the ordering clinician. The presence of a solitary kidney should not independently influence decision making regarding the risk of CI-AKI. In the setting of a recent AKI or if the eGFR is <30 mL/min, nephrotoxic medications should be withheld by the referring clinician, and volume expansion is recommended. Aside from the above considerations, when medically indicated, historical concerns over the potential renal toxicity of IV contrast should not alter contemporary evidence-based decision making. This is particularly relevant as we begin to replace nuclear stress testing with coronary CTA. A summary of these recommendations is provided in the table below.

Table: Summary of major ACR-NKF consensus statements on use of intravenous iodinated contrast media in patients with kidney disease

1. The terms CA-AKI or CI-AKI are recommended for use in clinical practice due to the large proportion of AKI events correlated with, but not necessarily caused by, contrast media administration.
2. The risk of CI-AKI from intravenous iodinated contrast media is lower than previously thought. Necessary contrast material–enhanced CT without a suitable alternative should not be avoided solely on the basis of CI-AKI risk.
3. CI-AKI risk should be determined primarily by using baseline CKD stage and AKI. Patients at high risk include those with recent AKI and those with eGFR ≤30 mL/min.
4. Kidney function screening is only indicated to identify patients at high risk for CI-AKI. Personal history of kidney disease (CKD, remote AKI, kidney surgery or ablation) is the strongest risk factor indicating the need for kidney function assessment.
5. Prophylaxis with intravenous normal saline is indicated for patients not undergoing dialysis who have eGFR ≤30 mL/min/1.73 m2 or a recent AKI. In individual high-risk circumstances, prophylaxis may be considered in patients with eGFR of 30–44 mL/min at the discretion of the ordering clinician.
6. Prophylaxis is not indicated for patients with stable eGFR greater than or equal to 45 mL/min.
7. The presence of a solitary kidney should not independently influence decision-making regarding the risk of CI-AKI.
8. When feasible, nephrotoxic medications should be withheld by the referring clinician in patients at high risk for CA-AKI.

1. Davenport MS, Perazella MA, Yee J, et al. Use of intravenous iodinated contrast media in patients with kidney disease: Consensus statements from the American College of Radiology and the National Kidney Foundation. *Radiology.* 2020;294(3):660-668. doi:10.1148/radiol.2019192094.

Perioperative gabapentinoids are associated with respiratory complications and do not decrease postoperative opioid use. They are now being used in a wide range of non-evidence-based scenarios.

Gabapentinoids are being used increasingly for osteoarthritis (OA) pain and chronic spinal radicular pain, both without an evidence base of support. A 2017 study[2] of acute and chronic sciatica looked at over 200 patients randomized to pregabalin up to 600 mg daily versus placebo for 8 weeks. Patients were then evaluated at 8 and 52 weeks. No significant between-group differences were observed with respect to the primary outcome of radicular pain reduction or any secondary outcome at either week 8 or week 52. A total of 227 adverse events were reported in the pregabalin group with only half that number in the placebo group. Dizziness was the most common, present in 40% of the pregabalin group. With respect to osteoarthritis, a British study[3] noted that prescriptions for gabapentinoids increased over 15-fold for OA from 2000 to 2015. Gabapentinoids are not even mentioned as a therapeutic option in the 2019 American College of Rheumatology osteoarthritis management guideline. Gabapentiniods are indicated for diabetic and postherpetic neuralgia, neuropathic pain post spinal cord injury and fibromyalgia. Given the paucity of evidence for other diagnoses and the very high incidence of side effects, they are not recommended for off-label use.

Gabapentinoids (gabapentin and pregabalin) are now being increasingly prescribed as part of perioperative pain-control protocols with an aim to reduce post-operative opioid use. However, the evidence to support this strategy is suboptimal with some data suggesting an increased risk of respiratory depression. Ohnuma and colleagues[4] assessed the dose-dependent effects of gabapentinoids on opioid consumption and pulmonary complications following total hip or knee replacement surgery. Using an existing database, the investigators identified 858,306 patients who underwent total hip or knee arthroplasty. Of those patients, 11% received gabapentin and 10.2% received pregabalin. Dosing for gabapentin was stratified into five groups, ranging from none to >1,050 mg per day, and dosing for pregabalin was stratified into four groups, ranging from none to >250 mg per day.

Receipt of gabapentin or pregabalin at any dose was associated with increased odds of respiratory complications. Compared to no exposure to gabapentinoids, gabapentin dosing >1,050 mg per day led to an odds ratio of 1.51 for respiratory complications; pregabalin dosing >250 mg per day led to an odds ratio of 1.81. Additionally, neither gabapentin nor pregabalin exposure reduced opioid consumption or decrease hospital length of stay.

Unless and until evidence of a beneficial effect of the perioperative use of this drug class has been established, they should not routinely be used in perioperative pain management. This is of concern as their use is becoming widespread in the United States. We can now add perioperative pain management to the list of indications for which gabapentinoids are ineffective. Gabapentiniods are only indicated for diabetic and postherpetic neuralgia, neuropathic pain post spinal cord injury and fibromyalgia. Once again, given the paucity of evidence for other diagnoses and the very high incidence of side effects, they are not recommended for off label use.

Behavioral therapy is effective, alone or combined with drug therapy, for men with symptoms of overactive bladder

The drug classes that treat overactive bladder symptoms include α-adrenergic receptor antagonists and antimuscarinic agents. In women, drug therapy combined with behavioral therapy is more effective than drug therapy alone. The effects of combined (drug plus behavioral) therapy for men, however, are not well understood. Burgio and colleagues[5] compared combined therapy versus individual drug or behavioral therapy among men with symptoms of overactive bladder.

In a multi-center clinical trial, 204 men (≥40 years of age) with urinary urgency and ≥9 voids per 24 hours were randomized to six weeks of behavioral therapy alone, drug therapy alone, or combined therapy. Drug therapy included sustained-release tolterodine (4 mg) plus tamsulosin (0.4 mg). After the initial six weeks, all groups were given combined therapy for an additional six weeks. Seven-day bladder diaries were completed before and after each treatment stage. The average number of voids per 24 hours decreased in all three treatment groups. Voiding frequencies were significantly lower in those who received combined therapy compared to those who received drug therapy alone, but not lower than those who received behavioral therapy alone. At 12 weeks, after all groups had received combined therapy, improvements in average voids were seen in all groups compared to baseline.

In elderly patients, potent anticholinergic therapies such as tolterodine have been shown to increase risk of dementia by 65%[6] and are discontinued by most patients within one year due to lack of effect or intolerable side effects[7]. Accordingly, behavioral therapy is optimal in treating men with overactive bladder symptoms. If a stepped approach in treatment is taken, consider starting with behavioral therapy and adding medications later for persistent symptoms. In all patients being treated with drugs for overactive bladder, deprescribing is an important part of management if drug response is suboptimal or side effects outweigh the benefit of treatment. http://www.camurology.org.uk/wp-content/uploads/pelvic-floor-exercises-male-27.pdf

2. Trial of pregabalin for acute and chronic sciatica. *N Engl J Med*. 2017;376(24):2396-2397. doi:10.1056/nejmc1705241.
3. Appleyard T, Ashworth J, Bedson J, Yu D, Peat G. Trends in gabapentinoid prescribing in patients with osteoarthritis: A United Kingdom national cohort study in primary care. *Osteoarthritis Cartilage*. 2019;27(10):1437-1444. doi:10.1016/j.joca.2019.06.008.
4. Ohnuma T, Raghunathan K, Moore S, et al. Dose-dependent association of gabapentinoids with pulmonary complications after total hip and knee arthroplasties. *J Bone Joint Surg Am*. 2020;102(3):221-229. doi:10.2106/jbjs.19.00889.
5. Burgio KL, Kraus SR, Johnson TM, et al. Effectiveness of combined behavioral and drug therapy for overactive bladder symptoms in men. *JAMA Intern Med*. 2020;180(3):411. doi:10.1001/jamainternmed.2019.6398.
6. Coupland CAC. Anticholinergic drug exposure and the risk of dementia. *JAMA Intern Med*. jamanetwork.com/journals/jamainternalmedicine/fullarticle/2736353. Published August 1, 2019. Accessed May 28, 2020.
7. Radomski SB. Drug persistence and adherence in the treatment of overactive bladder. *Can Urol Assoc J*. 2015;9(9-10):351. doi:10.5489/cuaj.3367.

CPAP versus standard of care in mild OSA

The evidence-based management of obstructive sleep apnea (OSA) was reviewed in the July/August 2019 edition of the Forum. It was then noted that when looking at the populations that served as the asymptomatic controls in multiple OSA studies, the apnea hypopnea index (AHI) increased with age. That meta-analysis in Lancet Respiratory Medicine[8] looked at over 5,200 healthy individuals who served as controls in sleep research studies and reported the sleep parameters derived from overnight polysomnography. At the age range from 18–64 years, the average AHI remained below 5 per hour, which is consistent with our definition of a normal AHI on our sleep study reports. However, in the age range of 65–80 years, the average AHI was 15, and over age 80, the average AHI was 30. There are good data that in patients with significant symptomatic OSA, treatment improves daytime sleepiness and fatigue, snoring and quality of life. Data, however, are lacking in the subset of patients with only mild OSA. Documenting improved outcomes in this group of patients is particularly important given the very high prevalence of sleep-disordered breathing with advancing age.

A recent study in The Lancet Respiratory Medicine[9] looked at the results of continuous positive airway pressure (CPAP) treatment in a population of patients with mild OSA. This was a multicenter, randomized trial that enrolled 233 patients between ages 18–80, with symptomatic but mild OSA (AHI 5–15). All patients had been referred to NHS sleep centers based on typical symptoms of OSA with an average Epworth Sleepiness Scale (ESS) score of 10, and all were studied using the ApneaLink home sleep study device. Patients were then randomized to sleep hygiene counseling versus an auto titrating CPAP unit and treated for three months. The outcomes favored CPAP therapy compared to the standard of care group, with a 10-point improvement in the SF-36 score. Most of the improvements were seen in the mental health components of the score, as opposed to the physical health components. There was also a modest improvement in the ESS score from 10 down to 7, with no ESS score change in the standard of care group. Compliance with CPAP use averaged four hours per night, a number that is consistent across multiple trials of CPAP therapy. At the end of the three months, 81% of the patients randomized to CPAP therapy chose to continue treatment.

This well-done trial confirms the benefit of CPAP treatment in patients with symptomatic, but mild OSA. It is important to note that although the AHI results fell into the mild category, the average ESS score of ten suggests that these patients scored in the "moderately symptomatic" range.

Also discussed in the prior Forum article were the data looking at CV risk and OSA. It is established that the other significant risks associated with OSA include an increased incidence of hypertension, and associated risks of cardiovascular disease and sleep related dysrhythmias. It is important to recognize however, that the data demonstrating a reduction of these risks through treatment of OSA is far more limited. There are data looking at hypertension control, and treatment of OSA has been associated with a small 4 mmHg improvement in systolic BP. However, there are not data showing reductions in cardiovascular risk with OSA treatment. Two important studies have looked at this.

- The first was a randomized trial of four years duration in 725 non-sleepy individuals with an AHI>20 and showed no reduction in the incidence of hypertension or cardiovascular events.[10]
- The second study was more compelling. It looked at a group of 2,700 patients with known CAD or stroke and moderate to severe OSA. The primary composite end point was death from cardiovascular causes, myocardial infarction, stroke or hospitalization for unstable angina, heart failure or transient ischemic attack. Patients were randomized to usual care or CPAP therapy and after 3.7 years, there was no reduction in CV events or improvement in mortality in the CPAP group.[11]

Examining this data in its totality suggests that treatment of OSA should be based upon symptoms and not coexistent disease. The United States Preventive Services Task Force (USPSTF) recently recognized this when it recommended against population screening for OSA in asymptomatic individuals. The important information added by this most recent study is that the subgroup of patients with significant symptoms but an only mildly abnormal AHI, are deserving of a trial of auto titrating CPAP therapy with continued treatment if symptomatic improvement is noted.

8. Boulos MI, Jairam T, Kendzerska T, Im J, Mekhael A, Murray BJ. Normal polysomnography parameters in healthy adults: A systematic review and meta-analysis. *Lancet Respir Med.* 2019;7(6):533-543. doi:10.1016/s2213-2600(19)30057-8.
9. Wimms AJ, Kelly JL, Turnbull CD, et al. Continuous positive airway pressure versus standard care for the treatment of people with mild obstructive sleep apnoea (MERGE): A multicentre, randomised controlled trial. *Lancet Respir Med.* 2020;8(4):349-358. doi:10.1016/s2213-2600(19)30402-3.
10. Barbé F, Durán-Cantolla J, Sánchez-De-La-Torre M, et al. Effect of continuous positive airway pressure on the incidence of hypertension and cardiovascular events in nonsleepy patients with obstructive sleep apnea. *JAMA.* 2012;307(20). doi:10.1001/jama.2012.4366.
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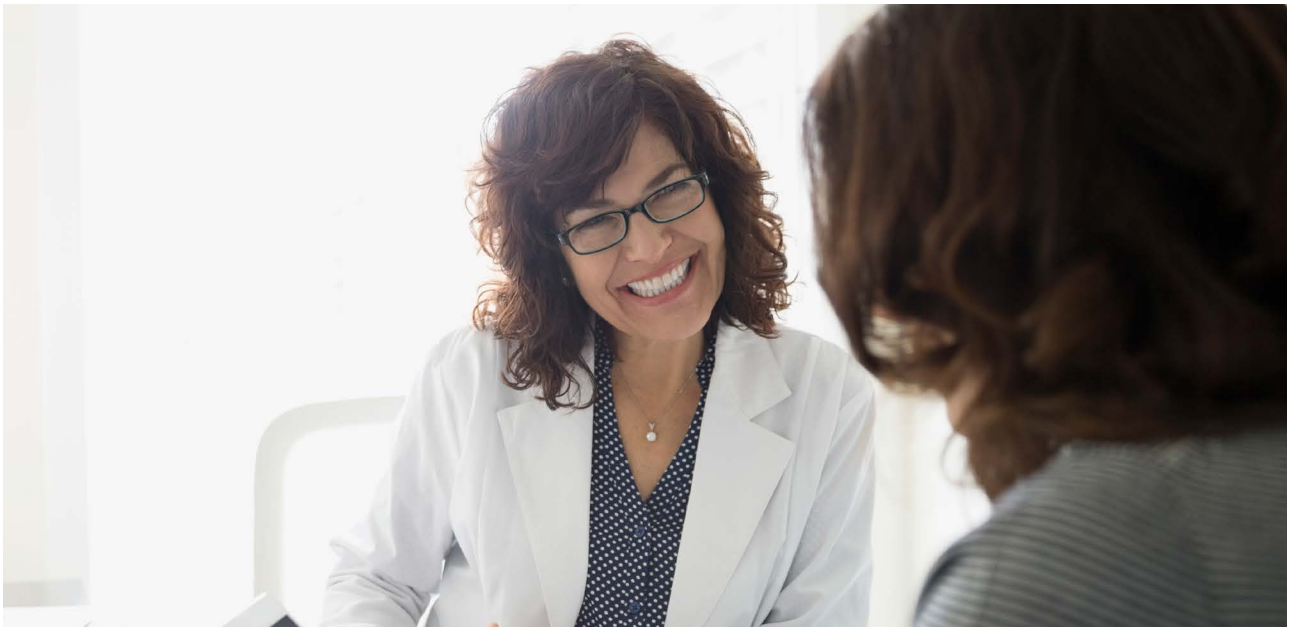

Is the echocardiogram of any value in the diagnosis of syncope in patients with a normal heart exam and ECG?

Syncope is estimated to account for 3% of all emergency room visits and up to 6% of hospital admissions. Lifetime prevalence of syncope is estimated to be 42%. Researchers at Abington Jefferson Hospital designed a retrospective chart review of patients admitted with syncope. They sought to understand the value of a transthoracic echocardiogram (TTE) in the setting of a normal physical exam and normal electrocardiogram (ECG).[12]

Researchers retrospectively reviewed charts of adult patients presenting with hospital admission for syncope over a two-year period. The review included 369 patients, of which 139 met all inclusion criteria.

Abnormal ECG defined	Abnormal TTE defined
• Abnormal axis • Ischemic changes • Conduction blocks including first degree, second degree, third degree blocks • Bi-fascicular blocks • Abnormal QTc • Left bundle branch block	• Ejection fraction <45% • Valvular abnormalities • Ventricular hypertrophy • Outflow tract obstruction • Pericardial effusion • Pulmonary hypertension

Of patients with an abnormal physical examination, 36% had an abnormal echocardiogram. In contrast, less than 1% of patients (1 of 120) with a normal physical exam had an abnormal echocardiogram. With respect to ECG abnormalities, the findings were similar. An abnormal echocardiogram was present in 23% of patients with an abnormal ECG, but in only 2% of patients with a normal ECG. A similar study[13] looked only at the value of the ECG in predicting an abnormal echocardiogram in patients presenting with syncope. Of 468 patients in the study, 210 (45%) had a normal ECG and underwent echocardiography. Excluding three patients with known severe aortic stenosis, only 4% had abnormal echocardiogram findings which were nondiagnostic and not related to the cause of syncope. Finally, a prospective observational study[14] showed that in 155 patients with unexplained syncope, routine echocardiography showed no abnormalities that established the cause of the syncope. Echocardiography was normal or nonrelevant in all patients with a negative cardiac history and a normal ECG.

The diagnostic value of the echocardiogram in patients presenting with syncope has been well studied with consistent findings over time. The use of an echocardiogram in the evaluation of syncope is not indicated in the presence of a normal physical examination of the heart and a normal ECG. It is highly overutilized in this setting.

12. Ghani AR, Ullah W, Abdullah HMA, et al. The role of echocardiography in diagnostic evaluation of patients with syncope-a retrospective analysis. *Am J Cardiovasc Dis.* 2019;9(5):78-83. Published 2019 Oct 15. Accessed May 28, 2020.
13. Chang N-L, Shah P, Bajaj S, Virk H, Bikkina M, Shamoon F. Diagnostic yield of echocardiography in syncope patients with normal ECG. *Cardiol Res Pract.* 2016;2016:1-7. doi:10.1155/2016/1251637.
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Kenneth Roy Cohen, MD, FACP | *Chief Medical Officer*

Dr. Kenneth Cohen is an experienced physician leader, practicing internist, and researcher who has attained national recognition for health care quality improvement. He has successfully developed and reported numerous clinical quality studies in primary care, including tobacco cessation, osteoporosis, asthma, diabetes, hypertension, and ischemic vascular disease. He was one of the founding physicians of New West Physicians, which is the largest primary care group practice in Colorado and now part of OptumCare. He has served as Chief Medical Officer since 1995. Dr. Cohen has received awards of recognition and distinction for teaching, including the Lutheran Medical Center Physician of the Year award in 2011. Under his stewardship New West Physicians was awarded the AMGA Acclaim award in 2015 and the Million Hearts Hypertension Champion Award in 2017. He is a Clinical Associate Professor of Medicine and Pharmacy at the University of Colorado School of Medicine. Dr. Cohen holds degrees from Dickinson College and Hahnemann University. He is a Fellow of the American College of Physicians and a member of the Phi Beta Kappa and Alpha Omega Alpha honor societies.

John Hitt, MD, MBA | *Senior Medical Director*

Dr. Hitt has been a physician executive for more than 25 years. Most recently he was the CMO of Ativa Medical a medical device startup company and an independent health care consultant. Previously, he was CMO at Maricopa Integrated Health System (MIHS) and a key member of the senior leadership team having responsibility for Medical Staff Services, Grants and Research, Academic Affairs, Risk Management, physician contracted services and the activity of Residency Program Directors, Clinical Department Chairs, and Medical Staff.

Dr. Hitt has over 25 years of experience in quality and performance improvement, clinical integration, academic and medical staff affairs. He served as the Chief Medical Quality Officer for Hennepin Health System, a premier Level 1 Adult and Pediatric Trauma Center. He was a physician leader for VHA (now Vizient). He was the national Medical Director for Disease Management at Caremark International and the VP of Medical Affairs at the University of Minnesota Hospital.

Dr. Hitt is a graduate of the University of Virginia where he played Division 1 soccer. He received his Medical Doctorate from the Medical College of Georgia in 1984 (AOA honors) and completed his Internal Medicine and Infectious Disease Fellowship training at the University of Minnesota Hospital and Clinics. Dr. Hitt completed his MBA at the Carlson School of Management at the University of Minnesota in 2003. He is the proud father of seven children.

Geoffrey Heyer, MD | *Senior Clinical Practice Performance Consultant*

Dr. Heyer is board certified in neurology with special certification in child neurology and in headache medicine. Prior to joining our team, Dr. Heyer was an associate professor of neurology and pediatrics at The Ohio State University and Columbia University Medical Center, specializing in autonomic disorders, headache, and pain management. He has published over 50 peer-reviewed research papers and numerous editorials, clinical reviews, and textbook chapters. He also co-authored a textbook on childhood stroke and cerebrovascular disorders.

Dr. Heyer received his medical degree from Columbia University, College of Physicians and Surgeons. He completed his neurology and child neurology residencies at Columbia-Presbyterian Medical Center. He has additional research training from the Mailman School of Public Health, Columbia University.

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