Radiology
September Podcast

Hi. This is Dr. David Bluemke in Madison, Wisconsin. I'm the Editor of the journal *Radiology*. This is part two of our September, 2019 podcast. The goal of these podcasts is to present a brief summary of key research in our field to keep you up-to-date. Today four articles to review. Safety of 7-T MRI, using MRI to evaluate patients with atrial fibrillation, iodine contrast and renal injury, and dual-energy CT for head trauma.

Safety Considerations of 7-T MRI in Clinical Practice

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Let's start with a review article about MRI safety. The title is *Safety Considerations of 7-T MRI in Clinical Practice.* The first author Dr. Michael Hoff from the University of Washington in Seattle. The senior author Dr. Emanuel Kanal at the University of Pittsburgh. 7-T clinical MRI is now approved for routine use. Most of us do not own a 7-T MRI scanner. But the first topic that comes to my mind is safety. Not too long ago we thought that an artificial heart valve would be torn out of a patient's chest even at 1.5 T. Well, I'm pretty sure it's not going to happen at 7 T either but a good time to review the topic. Are we reaching a limit on increasing magnetic field strength? A brief history of 7-T MRI. In the United States there are more than 30 MRI scanners operating at 7-T field strength or higher. In Europe, more than 20 scanners exist at this field strength. Same for China, Asia, Japan, Korea, and Australia. Multiple 7-T scanners already in place. The highest field strength MRI scanners in the United States have been consistently located at the University of Minnesota, a research human MRI operating at 11.7 Tesla. These ultra-high-field strength magnets become a major technical challenge to operate. They are supported by a whole team of engineers. They can be a technical tour de force or a disaster. During my tenure at NIH, the 11.7-T MRI scanner essentially had a meltdown. The magnet overheated and was permanently damaged. It took about two years to get a replacement magnet, only arriving at NIH in April of this year. That magnet delivery, 51 tons, 35,000 liters of helium, 380 tons of steel added to the building at NIH for magnetic shielding. Why 7-T MRI in the first place? The main reason: Better imaging of the brain. Signal-to-noise is approximately proportional to field strength. So in theory you get about twice as much signal at 7 T versus 3 Tesla. The 7-T images of the brain are dramatic. Similar detail to a brain at autopsy without the need for patient death. The goal is resolution of less than 1 millimeter to resolve individual layers of the brain cortex. The issue of the day in brain MRI is connectivity, mapping the entire connectome of the brain. The idea of the connectome in theory is to map the individual connections of every neuron and all done better at 7 T. The challenge: The human cerebral cortex alone contains on the order of 10 billion neurons linked by 100 trillion synaptic connections. By comparison the human genome has a mere 3 billion base pairs. As early as the year 2003, the U.S. Food and Drug Administration or FDA approved MRI at up to 8 Tesla as nonsignificant risk for neonatal patients. In 2017, Siemens received approval in Europe for a clinical whole-body 7-T MRI scanner. In the United States approval for the same 7-T MRI was also granted in 2017. I had a chance to talk with the head of Siemens MRI Division about the time of the 7-T approval. I was interested in the cost of the machine. I reminded him of the rule of thumb. We used to say MRI machines cost about 1 million dollars per Tesla. I mentioned that to the Siemens executive. He looked at me with a slight grin and said, "No. No. It's a bit more than that at 7 T." Back to the topic of safety. What happens when we put humans in the 7-T scanner? There are four major reports about the effects on humans involving more than 3,000 research participants. Major effects to-date: Number 1: Vertigo. The patient lies on the MRI table and the table is moved into the bore of the 7-T scanner. Almost 40 percent of patients experience dizziness or vertigo just from moving the table. At one site the rate was 60 percent of patients. The problem seems to be the ionic fluid in the vestibular apparatus of the inner ear. Ionic fluid responds to a magnetic field causing motion of the endolymph then dizziness and nausea. Some reports indicate this could persist for up to 30 minutes after leaving the scanner. Number 2: Effect on vision. Changing magnetic fields can induce current in the retina. This stimulates the optic nerve. The patient sees flashing lights. These visual effects of flashing lights relate to the change in magnetic field per unit time. We call that dB/dt. The B is magnetic field. The t is for time. dB/dt. Effects on vision seem to be infrequent, 1 to 2 percent of patients. Number 3: Effect on taste. The electrogustatory effect. The patient experiences a metallic taste. One report found half of patients noticed this metallic taste. The reasons are not known. One possibility: Metallic dental fillings could be affected. In a prior podcast, I discussed the effect of 7-T MRI on dental fillings. For mercury fillings, the researchers found free mercury in the saliva at 7 T but not at 1.5 T. But these were fresh new fillings. Number 4: Peripheral nerve stimulation. We already know about this from 3 T. The effect is related to the change in magnetic field not the absolute field strength. Right now those gradient changes are greater at 3 T than at 7 T. Number 5: Effect on the heart. Blood contains electrically-charged ions. In theory, a strong magnetic field will induce a force in the blood opposite to that of the direction of normal blood flow. At 7 T, there have been reports of sinus tachycardia, rapid heartrate. Although this seems to be not a major concern patients with heart failure have not yet been reported. Number 6: Implanted devices at 7 T. Begin with the basic process of making an image with MRI. The patient is magnetized in the bore of the magnet. This means that a very small proportion of protons, hydrogen atoms, are lined up like little bar magnets north pole to the north pole of the magnet. That's the static magnetic field. In order to get an image, we transmit radiofrequency energy into the patient. This is usually done with a big antenna that surrounds the patient, the body coil. The RF energy disturbs the equilibrium of the bar magnets in the patient. Then we turn off the radio energy and the protons relax to their original position before we started. As the protons relax back to the starting point, they emit a small radio signal. The radio signal from the protons is picked up by the body coil antenna or a smaller coil like the knee coil can be used. The electrical signal is processed to form an image. How are implants and devices affected by this process? We transmit a lot of energy into the patient in order for the patient to give us back a very small signal. That radiofrequency energy can affect implants and devices in the human body. Already at 7 T, about 300 devices have been tested for safety. But for 1.5 and 3 T, we have safety information on more than 6,000 devices. Still a way to go for 7 T. What happens to a wire like a pacemaker lead or other implant in the body? In some cases, the wire or implant can absorb radiofrequency energy from the transmit coil. If the radio energy gets absorbed by the device the device or wire acts like a little wire in your toaster, red hot, at least in the toaster. Under what conditions can absorption of radio energy occur? The answer: When the device or wire has a size that

is one-half of the wavelength of the RF energy. Here are the numbers: At 1.5 T, a wire at multiples of 26 centimeters could potentially absorb the energy. At 3 T, the number is 13 centimeters. At 7 T, it's really quite short. A device or wire at multiples of about 5 centimeter length could absorb the RF energy. You can see why our physicists get excited about this. Lots of devices about that size, lots of possibility for making a toaster wire in the body. Okay, now for the good news. At 1.5 T, you usually use the body coil to transmit the energy to the patient. The entire patient is irradiated with radiofrequency energy, every device, every staple, every wire. But at 7 T, there is no body coil transmitter. In the clinical 7-T system the only transmission coils are the head coil and the knee coil. The RF energy is really directed at the knee or the head only. This should help safety. Devices and wires in the chest or abdomen are exposed to a lot less radio energy. That's a brief summary. Many of these same safety issues apply to 3 T and 1.5 T. Next, onto our research articles for September

Left Atrial Fibrosis Assessed with Cardiac MRI in Patients with Paroxysmal and Those with Persistent Atrial Fibrillation

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The next topic is about atrial fibrillation, a very common disease. The title is *Left Atrial Fibrosis Assessed with Cardiac MRI in Patients with Paroxysmal and Persistent Atrial Fibrillation.* The research is from Radiology and Cardiology Departments at Korea University Anam Hospital. The first author is Dr. Dong Kyu Lee. Background: You've probably seen advertisements on television about treatments for atrial fibrillation. About 6 million people in the United States are estimated to have this condition. That's about 9 percent of people age 65 and older. Almost an epidemic. Atrial fibrillation or AFib can result in blood clots forming in the heart in the left atrial appendage. Stroke can result. Atrial fibrillation is a rapid twitching contraction of the atria. The atria are supposed to contract in a coordinated manner just like the ventricles to pump blood. But atrial contraction is completely ineffective in AFib. The blood stagnates in the left atrium and in the left atrial appendage in particular. Blood can clot because the motion of the blood is so slow. If you are reading any CT of the chest in an older patient, you want to check the left atrial appendage routinely for evidence of clot. Why does AFib occur? Here is a scenario using the example of pizza and salty food. You really like pizza. A single slice of pizza contains one-quarter of your recommended daily dose of salt. Maybe you go for an entire half of pizza, maybe the whole thing. Way too much salt. Your blood pressure increases due to salt. The effect of chronically increased blood pressure is to stretch the heart especially the weak thin-walled atrium. As the atria dilate there are stretch receptors that notice. These receptors fire in an irregular manner. You might not even notice but your pulse becomes irregular or too fast. You could feel faint or weak with exercise. If this abnormal left atrial stretch continues the left atrium begins to dilate. How does the heart respond to chronic stretch? The heart counteracts chronic dilatation by strengthening itself. It creates a scar, collagen in the heart. But heart is simply not supposed to have scar. The heart is nearly all muscle, no scar. What happens next? The pacemaker for your heart is in the sinoatrial node. The sinoatrial node is in the right atrium. The SA node sends out an electrical pulse that is supposed to be conducted down to the ventricles. But now there is all of this fibrosis in the heart. The normal pathway down to the ventricle is interrupted by the scar, the fibrosis. The electrical signal starts to find zig-zag patterns all around the scar trying to make it down to the ventricle. When the electrical zig-zag pattern becomes too much it can form little circles of electrical current in the wall of the atria. So one pulse from the SA node keeps circulating around causing the atria to try to contract really quickly. This electrical pulse that goes around and around in circles is called a reentrant circuit. Every time the circle hits the atria it tries to contract eventually going onto atrial fibrillation. You might know a younger person with atrial fibrillation perhaps in their 30s or 40s. Usually for young people the atrial fibrillation does not last long. It starts at random times and then stops by itself. The person feels ill at ease or can feel faint while this happens. The term for this condition is paroxysmal atrial fibrillation. Now an older person with AFib, maybe 70 years old, their AFib can become almost permanent. It does not come and go. This is called persistent AFib and is much more dangerous. These are the patients who get treatment. The original treatment for AFib was open heart surgery. The procedure is still done today but not as often. It is called the maze, M-A-Z-E procedure. Hard for me to believe when I first heard this. The surgeon makes small incisions in the inside wall of the left atria. Those incisions also cause a scar but the linear maze scar lines prevent the electrical currents from making those circles. It prevents the reentrant arrhythmia in the atria. Today, heart surgery is hardly used for AFib, only for the most difficult cases. Instead the cardiac electrophysiologist puts a catheter into the left atrium. She uses a small wire loop and makes burns in the atrial wall. The burns form a scar and prevent electrical conduction. The burns are made at the openings or ostia of the pulmonary veins as they enter the left atrium. There are two pulmonary veins from the right lung, two from the left. These bring oxygenated blood from the lungs to the left atria. So that's the paradox. Atrial fibrillation is caused by scar in the enlarged left atrium, and the treatment is to make more scars in an organized pattern to prevent the reentrant electrical circuits from continuing. Where does MRI fit in? As you know, the number one reason for doing MRI of the heart is to find scar. But that's in the left ventricle. Purpose: To use MRI to determine the pattern of scar in the left atrium in patients with atrial fibrillation. Methods: This was a retrospective review of nearly 200 patients. The patients had an MRI of the heart prior to catheter ablation of the left atrium for AFib. The method to find the scar in the left atrium: Similar MRI pulse sequences as used for the left ventricle but we need thinner 1 to 2 millimeter slices. Inject gadolinium, usually a double dose of gad. Wait for 15 minutes. Then acquire late gadolinium images or LGE MRI. Scar in the left atrial wall is bright. The myocardial tissue is suppressed made to be dark due to an inversion recovery pulse. The authors measured the size and location of the left atrial scar. They also measured the size of the left atrium. Results: Of the 200 patients, about 40 percent had the less severe form of AFib, intermittent or paroxysmal AFib. The other 60 percent of patients had AFib all the time, persistent AFib. Of all of the 200 patients, MRI found scar in 60 percent of cases. For patients with the most severe persistent AFib there was about 50 percent more scar in the heart. There was an interesting pattern to the scar location in the left atrium. It was mostly in the posterior wall and mostly around the left inferior pulmonary vein. Large size of the left atrium and the amount of scar were both more common in persistent AFib as expected. In the final analysis, scar around the left inferior pulmonary vein was four times more common in patients with persistent AFib compared to paroxysmal AFib. Conclusions: The use of MRI to find scar in AFib has been going on for about ten years. Most of us were quite skeptical that it worked at all. The wall of the left atrium is very thin, only about 2 millimeters, but researchers at the University of Utah led the way. Their MRI technique was beautiful. The scar pattern had proof in animal models, at pathology, and in the electrophysiology lab. The evidence is adding up. MRI can not only find scar in the left ventricle, it is poised to have a greater role for patients with atrial fibrillation, a huge number of these patients as the population ages. In this study more scar on MRI was associated with worse AFib. There are four pulmonary veins entering the left atrium but the worst scar was on the left side coming from the lower left lung. A very nice MRI study by this research group from Korea.

Kidney Injury after Intravenous versus Intra-arterial Contrast Agent in Patients Suspected of Having Coronary Artery Disease: A Randomized Trial

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The next article is about renal injury from iodinated contrast. The title, *Kidney Injury after Intravenous versus Intra-arterial Contrast Agent in Patients Suspected of Having Coronary Artery Disease: A Randomized Trial.* The first author Dr. Eva Schonenberger. The senior author Dr. Marc Dewey. The authors are from Charite University Hospital in Berlin, Germany. Background: Thirty million doses of iodine contrast agents are given per year with CT and another 2 million for cardiac catheterization. Let's review the controversy of iodinated contrast agents in relationship to acute renal injury. First, the definition of contrast-induced acute renal injury. It is an increase in serum creatinine by more than 25 percent or 0.5 milligrams per deciliter. The decrease in renal function occurs within 3 days or 72 hours. Second, the next question, how often does this occur? The controversy: Some researchers suggest that iodine contrast is not even the real cause of acute renal injury. The background: Many early research reports said that acute renal injury occurred in 4 to 20 percent of patients after iodinated contrast. Then, in 2008, Dr. Jeffrey Newhouse at Columbia University published an interesting study in AJR of 32,000 patients. Half of these met the criteria for acute kidney injury but none of these patients had exposure to iodinated contrast. This raises an interesting question. Suppose I want to see if an iodinated contrast agent causes acute renal injury? I measure GFR before the CT scan, then give iodine, then measure GFR again. The creatinine goes from 1.0 to 1.5 meeting the definition of acute renal injury. Right? No. Dr. Newhouse pointed out that's not right. If I study only those patients who receive a contrast agent I have no control group. I do not know what happens to those patients who do not receive iodine. We need a similar group of patients who had baseline GFR, then no iodine, perhaps a non-contrast CT, then measure GFR again. A control group has been missing. Next major development, in 2013, Dr. Matthew Davenport, a major study published in *Radiology*, 17,000 patients using a technique called propensity matching. Propensity matching creates an artificial control group to compare those with versus without iodine contrast reaction. Dr. Davenport's results: He saw no evidence of acute renal injury unless renal function was lower than 45 mL per minute. Renal injury is more common at GFR less than 30 mL per minute. More evidence: 2014, Dr. Robert McDonald, Mayo Clinic, also published in *Radiology*, again, a propensity analysis was done to create a control group, 21,000 patients. Here, no effect of iodine from contrast injection. No greater risk of acute renal injury. No need for dialysis or death that was excessive in patients who had iodine injection versus those who did not. More data followed, also from Mayo Clinic, published again in *Radiology*, 12,000 patients, mostly inpatients. Acute kidney injury happened more frequently if the GFR was lower but no difference in acute kidney injury whether or not the patient had an iodine injection. The authors conclude the risk of acute kidney injury is independent of contrast agent exposure even if the GFR is less than 30 mL per minute. Conflicting confusing data in the literature. Some authors suggesting that after you control for comorbidities there is no such thing as acute kidney injury from iodine contrast for CT. Another major study. Renal injury is more likely if GFR is less than 30 mL per minute. The common factor: If your patient has poor renal function that patient is at risk for acute renal injury. In a meta-analysis of studies that include a control group the risk of iodine appears much less than we were taught, probably less than 1 percent risk. These results then are the basis for recommendations in the American College of Radiology Contrast Media Manual. First, the terminology. The best term is post-contrast acute kidney injury. Decreased renal function after CT could be from iodine but could be due to other treatments as well, chemotherapy drugs, antibiotics, dehydration. To quote the final ACR recommendation, "If a threshold for contrast-induced nephropathy risk is used at all, 30 mL per minute seems to be the one with the greatest level of evidence." One more twist. How do we explain why some very careful researchers found renal injury but others did not even when a control group is used? Some research has zeroed in on intra-arterial contrast for cardiac cath versus contrast given intravenously for CT. The differences: Exposure to high concentrations of iodine for the coronary arteries. But that can't really be for the kidneys, right? Coronary arteries drain to the coronary veins, then where? To the coronary sinus, to the right atrium just like venous injection. One other possibility. Micro emboli to the kidneys from a catheter in the aorta. Purpose: To compare the rate of acute kidney injury using intra-arterial versus intravenous administration of iodine contrast. Methods: This was a prospective clinical trial of 320 individuals. All of the patients had suspected coronary artery disease. Patients were randomized to either have a coronary CT or have a standard cardiac catheterization. So half of the patients had intravenous contrast with CT, half had intra-arterial contrast. The average age was 60 years. The two groups were well matched for risk factors. Results: For patients who had a cardiac cath with intra-arterial iodine injection, 13 percent with acute renal injury. For those who had CT, the rate of acute renal injury was less than half, about 6 percent. That's a relative risk of 2.4 times greater for renal injury if there was a cardiac cath versus CT scan. The patients were followed for about two years. If the patient had acute kidney injury from iodine contrast that patient had 12 times greater risk of chronic kidney disease at two years. Patients who had a CT scan had slightly less volume of iodine contrast than cardiac cath patients, 66 mL for CT, and 78 mL on average for cardiac cath. Conclusions: We continue to search for answers as to why some patients have decreased renal function after CT. One major study, 5.5 percent of patients who had a CT scan with iodine contrast had acute renal injury, but add a control group. Propensity matched hospitalized patients without a CT scan, 5.6 percent of those patients also had acute kidney injury. Today's research study, well done, prospective, randomized clinical trial, about 2 times less likelihood of acute renal injury after CT than after arterial injection of contrast with cardiac catheterization. Final comment: The search for risk factors and treatments for acute renal injury continues. Lots of evidence about dehydration resulting in greater likelihood of iodine contrast and renal failure. About half of major reports find that the risk of iodine is worse if GFR is less than 30 mL per minute.

Iodine-based Dual-Energy CT of Traumatic Hemorrhagic Contusions: Relationship to In-Hospital Mortality and Short-term Outcome

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Final article for today. Dual-energy CT and head trauma. The title is *Iodine-based Dual-Energy CT of Traumatic Hemorrhagic Contusions: Relationship to In-Hospital Mortality and Short-term Outcome.* The first author Uttam Bodanapally. The senior author Thorsten Fleiter. The authors are from the world-famous Shock Trauma Hospital in Baltimore, Maryland. Background: Dr. Chris Hess is Chair of the Department of Radiology at the University of California of San Francisco and on our Editorial Board. Together with Dr. Jason Talbott, Associate Professor of Radiology at UCSF, they have written a nice editorial for this article. A quote from parts of that editorial for my discussion. "Traumatic brain injury or TBI affects 50 to 60 million patients per year worldwide. There are four types of TBI. Hemorrhagic contusion, subarachnoid hemorrhage, hematomas such as subdural and epidural hematoma, and diffuse axonal injury. Diffuse axonal injury is seen most commonly on MRI. Of these, brain contusions are common and substantial contributors to morbidity and mortality. Contusions most often result from direct impact of the brain on adjacent rigid structures; thus they tend to originate superficially in gray matter near the skull. The irregular surfaces of the anterior and middle skull base make the anterior and inferior portions of the frontal and temporal lobes vulnerable to contusion injury. Hemorrhagic brain contusion on CT is irreversible brain injury. However, there is a more recently defined phenomenon called hemorrhagic progression of contusion. The normal-appearing surrounding areas of the brain at CT undergo hemorrhagic conversion with expansion of the contusion area at follow-up CT." What is the reason for the hemorrhagic progression of contusion or HPC? At the core of the hemorrhage, there is fragmentation of capillaries leading to the immediate leak of blood. But around the hematoma the so-called penumbra the capillaries are intact at first. Over a matter of hours, there is adverse endothelial response in the penumbra of brain matter around the contusion. The surrounding areas also undergo capillary fragmentation. The overall appearance on conventional CT is an expansion of the contusion. Purpose: To use dual-energy CT to quantify iodine leak over time in the penumbra surrounding the initial focus of brain contusion. Methods: Patients have a non-contrast brain CT for trauma evaluation. But patients with whole-body trauma also have body CT performed with iodine contrast. The authors identified patients who had a brain contusion on the initial emergency department CT scan. Sixty-five patients had a follow-up CT an average of six hours later in conjunction with contrast-enhanced whole-body CT. The problem with single-energy CT. You see an initial hematoma, say 2 centimeters. Six hours later you see the hematoma has grown perhaps 5 centimeters, so there is a large leaky area around the initial contusion that grew. But is this just an iodine leak? Or is it frank hemorrhage? With single energy you cannot tell. We think of the penumbra surrounding the hemorrhagic core of the contusion as containing leaky microvasculature but without frank hemorrhage. On single-energy CT, both the penumbra and the central hemorrhage from the contusion are dense on CT scans at 120 keV. The next step: Perform monoenergetic dual-energy CT reconstruction. At a high keV of 190, the iodine is not visualized. Why? Recall that the iodine k-edge is at 33 keV. A reconstruction at 190 keV is far from the 33 keV iodine k-edge. That means very little contribution of iodine to the CT density. Therefore, at 190 keV, the extravasated iodine does not confound the measurement of actual hemorrhage in the middle of the contusion. At 190 keV, we see only the dense blood at the middle of the contusion not the iodine leak. The authors saw a large hematoma at the standard energy of 120 keV iodine plus blood and subtracted the smaller central core of hemorrhage. They looked at separate relationships of the infarct core and the penumbra to patient outcome after trauma. Results: The greater the iodine leak after six hours the worse the patient did. The amount of iodine in the penumbra at six hours was the best imaging predictor of mortality as well as long-term disability for the patient. Using dual-energy CT, all brain contusions in this study were associated with iodine leak. So using only single-energy CT you cannot determine if you have a true expanding hematoma versus leaking capillaries around the central hematoma. Conclusions: We think of patient outcome as related to the size and severity of brain contusion. But dual-energy CT allows the two components of the brain contusion to be separated, the central contusion and the surrounding penumbra of brain tissue with leaking capillaries. In this study, dual energy was superior to single-energy CT measures of patient outcome in the hospital as well as longer term disability. The authors recognized that perfusion CT and contrast MRI could also be used to detect microvascular disruption. Still, dual-energy CT is becoming widely available. In the CT protocol, the authors combined their head CT with whole-body CT done with iodinated contrast looking for other injuries from polytrauma. The authors of this study open the door for using CT to quantitatively assess injury due to traumatic contusion and hemorrhagic progression of contusion. CT factors that describe the contusion are still not as predictive of in-hospital mortality and disability compared to the initial Glasgow Coma Scale rating. But this is the first step towards more accurate and routine hematoma assessment using dual-energy CT after head trauma.

That concludes this week's articles. I hope these articles have been useful to you. I hope you have a very good rest of your week. This is Dr. David Bluemke for the journal *Radiology*.