

**BODY MR IMAGING: BASIC PRINCIPLES, GENERAL RULES,
AND KEY FACTS**

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Introduction

Welcome to the MR body imaging service!

We hope that you learn a lot and enjoy your time on this rotation.

Although body MR is exciting, challenging, and fun, it can also be intimidating. There are seemingly innumerable variables, technical parameters, options, and terminology to learn. In our opinion, body MR is the single most intellectually challenging discipline in diagnostic imaging.

The following guide discusses some basic principles of body MR imaging and is intended as a supplement to the handout on MR imaging protocols. We hope that this guide will help make MR imaging less mysterious and intimidating and more fun.

The guide is a work in progress. It is intended to evolve, become more complete, and hopefully to improve as experience is accrued. We welcome and encourage you to contribute to this guide and to the protocols handout by giving us feedback, constructive criticism, and suggestions. Please let us know what sections are too long, too brief, too confusing, etc. What sections would you like us to add? What sections are redundant or unnecessary? What protocols are inadequate, suboptimal, or incomplete?

If you have the motivation and the interest, help us draft a new or better protocol or write your own section to this guide.

We also want to emphasize that the protocols listed in the protocols handout are meant only as a starting point. We recommend tailoring protocols to individual cases to best answer the relevant diagnostic question(s).

Finally, do not hesitate to contact the MR imaging faculty if you need help for whatever reason.

Overview: MR vs. the competition

The major competition between MR and ultrasound (US) is availability and cost, while the major competition with CT is access to scanners. MR imaging applications are gaining greater acceptance due to improved image quality of the fast imaging sequences, the tissue characterization potential that exceeds that of CT and US, the availability of off-axis imaging, the ease of angiography, the safety of the Gd-chelates even in renal failure, and the availability of liver specific contrast agents, iron oxide (Feridex, Berlex Laboratories) and Mn-DPDP (Teslascan, Nycomed-Amersham). While US can image in any plane, it has a limited field of view, is hindered by gas, bone and excessive fat, and it lacks the tissue specificity afforded by MRI. Also, although ultrasound demonstrates blood vessels reasonably and gives information on their flow dynamics, MR depicts all vessels. On MR, vessels typically exhibit signal void on spin echo sequences and bright signal on gradient-echo sequences. Moreover, flow-induced phase changes can be controlled and manipulated to obtain accurate flow velocity in any direction.

CT detects differences in electron, or mass, density (one parameter). US detects differences in acoustic impedance between adjacent tissues as well as the heterogeneity of tissue structure (two parameters). MR detects differences in hydrogen density, T1 and T2 relaxation times, chemical shift induced by fat and water, and flow (five parameters). The tissue characterization potential is therefore greatest for MR.

How to Protocol a Case

General Rules and Principles

The single most important aspect of writing an appropriate protocol is obtaining a pertinent history. This may mean reviewing clinical data on the PCIS system, calling the clinician, and interviewing the patient. If you know and understand the relevant clinical question(s), you will be better able to protocol a case and interpret the findings.

The second most important aspect, often necessary to satisfy the first, is to review pertinent prior imaging studies. In general, use the same protocol as before unless the previous protocol left unanswered questions that could be better evaluated with a modified protocol.

Third, if you have any questions, please page the MR attending of the day. It is our responsibility to help you learn and deliver optimal patient care.

Fourth, more is not always more. Sometimes more is less. Resist the temptation to simply add extra sequences because you are unsure of what to do next. Again, when in doubt, call your attending.

Once you have obtained appropriate clinical information, reviewed old studies, and, if necessary, discussed the case with the attending, you are ready to write a

protocol. In writing a protocol, you should consider at least the following six sets of variables:

- a) Patient preparation.
- b) Patient position (prone or supine).
- c) Coil type and location.
- d) Contrast type and dose.
- e) Imaging sequences, planes, relative FOV (eg, small or large), and, depending on the case, other parameters.
- f) If you are planning to do dynamic imaging with gadolinium, decide whether you want to do a timing run or a best guess for determining scan delay.

These are summarized below and enumerated specifically in the protocols handout.

1. Patient preparation. Usually, no special preparation is necessary. Some cases do require special preparation, however. These include MRCP, female pelvis, breast MR, and prostate cases and are discussed later.

2. Patient position. For the vast majority of cases, patients are positioned supine. (Therefore, supine is the default position). If it is essential to minimize the AP excursion of the chest or abdominal wall during breathing (eg, some RV dysplasia and superficial soft tissue cases), you may want to position the patient prone.

3. Coil type and location. For most abdominal and pelvic imaging in adults, use the body coil and/or the phase array coil. The major advantage of the phase array coil is that it increases signal to noise for thin patients. The major disadvantages of the phase array coil are that it is ineffective in large patients, it is prone to severe respiratory and other motion artifacts, and it has limited craniocaudal coverage (approximately 30 cm). Keeping these advantages and disadvantages in mind, we recommend the following:

1. If the patient is large (ie, > 20 cm in AP), use the body coil only, and don't bother with the phase array coil. Use your judgment. One exception is the pregnant female. If the scan is done to evaluate the fetus, then the phase array coil may be appropriate depending on the location of the fetus relative to the mother's skin surface. If the purpose is to evaluate the mother, the body coil would probably be better. Again, use your judgment.
2. If the patient is thin (ie, < 20 cm in AP), attempt to use the phase array coil by placing the coil over the region of interest. At UCSD, we reposition the coil during the examination as appropriate (eg, over the abdomen when imaging the abdomen, over the pelvis when imaging the pelvis).

3. After the localizer is obtained, check the images to make sure the coil is in the correct position and that the images are not marred by artifact. If the coil is not correctly positioned, either reposition it or switch to the body coil. If motion or other artifact is significant (usually not a problem in the pelvis, but a frequent problem in the abdomen), switch to the body coil.

For other indications, use other coils:

Scrotum:	surface coil
Brachial plexus:	neurovascular coil
Prostate:	simultaneously use the anterior elements of the phase array coil (turn off posterior elements) and endorectal coil.
Rectal cancer:	ditto
Infants:	knee coil
Breast cancer:	breast coil
Superficial ST's	surface coil

Coils are described in greater detail in a subsequent section.

4. Contrast type and dose. For all vascular cases and many body cases, you will need to use an extracellular gadolinium chelate. At UCSD, we administer gadolinium as a bolus using a power injector followed by a 20 ml saline chaser. For all vascular cases and for a subset of non-vascular cases, use a “double” dose of gadolinium (40 ml for an adult) at a relatively rapid rate (3 ml/sec). For non-vascular cases, a “single” dose (20 ml for an adult) at a rate of 2 ml/sec usually suffices, unless you know from a previous scan that a particular patient requires a double dose to achieve a satisfactory study.

In almost all cases in which gadolinium is used, dynamic imaging should be done. In dynamic imaging, sequences are obtained in quick succession beginning with peak arterial phase. Dynamic imaging is critical for arterial vascular cases and is also helpful for detecting and characterizing neoplastic processes. When imaging a large body area (eg, a combined abdomen and pelvis study) do the dynamic imaging over the region in which the information will be most beneficial. For example, in a postoperative patient with rectal cancer, do dynamic imaging through the pelvis if evaluation of soft tissue in the surgical bed is desired and do dynamic imaging through the liver if evaluation of hepatic lesions is desired.

As discussed further in subsequent sections, Feridex and/or Teslascan can be used in selective liver cases. Use the standard dose recommended by the manufacturer when using these agents. (Feridex: 0.05mL/kg body weight (~1mL/40 lbs) diluted in 100 mL of D5W, infused over 30-60 minutes; Teslascan: 0.1 mL/kg (~0.5 mL/10 lbs) infused relatively slowly over one minute, with maximum dose of 15 mL).

More on contrast agents later.

5. Imaging sequences, planes, FOV, and other parameters.

Sequences:

Pre-gadolinium: Usually, you will perform both T1w and T2w sequences before contrast administration, often in more than one imaging plane. Many options are available.

T1w. At UCSD the two most commonly used T1w sequences for body imaging are (1) T1w turbo spin echo (TSE) and (2) T1w GE (2d FLASH). The T1w TSE sequence usually takes at least 2 minutes to acquire and is therefore obtained while the patient is breathing quietly. The GE sequence takes only 15-25 seconds and is obtained while the patient breathholds. As a general rule, the TSE images show better anatomic detail than the GE images and are superior when breathing motion is not much of a problem (eg, pelvis, prostate, rectum, scrotum). The GE sequences are better, however, when breathing motion is a problem (eg, abdomen, liver, kidneys). Remember that the GE sequences can be obtained out of phase (TE ~ 2.2 msec) or in phase (TE ~ 4.4 msec). More on this later. **NOTE:** If the patient is uncooperative or extremely dyspneic, a 2d turbo FLASH can be attempted in addition to or instead of the 2d FLASH for T1w imaging.

T2w. The four most commonly used T2w sequences for body imaging are (1) T2w TSE, (2) modified breath-held T2w TSE, (3) HASTE, and (4) FISP. The T2w TSE sequence, similar to the T1w TSE sequence, takes over two minutes to acquire and is therefore obtained while the patient is breathing. The modified breath-held T2w TSE is similar to the standard T2w TSE but is modified (including a longer echo train length) so that it takes only 20-25 seconds to acquire and is done as a single breathhold. The modified TSE sequence provides less lesion-to-parenchyma contrast than the standard TSE sequence but does not suffer from motion artifact. Therefore, we prefer the modified sequence when respiratory motion needs to be eliminated (eg, liver) but we prefer the standard sequence when respiratory motion is not a problem (eg, pelvis, scrotum, prostate, rectum). If the patient is extremely dyspneic or has a lot of ascites, a HASTE sequence may be preferable to the modified breath-held TSE sequence, as each image takes only ~1 second to acquire. HASTE images are also ideal for evaluating fluid-filled structures such as bile ducts, pancreatic ducts, collecting systems, and ureters and are routinely obtained as preliminary scans. FISP sequences can also be helpful when ultrafast T2w imaging is desired.

Pre-Gadolinium fat-saturated T1w images. Pre-gadolinium fat saturated T1w images (T1w 2D FLASH with fat sat) are essential in all chest and superficial soft tissue cases in which you decide to give gadolinium. The reason is that air-soft tissue interfaces in the chest (lung-mediastinum) and superficial soft tissues (skin-outside air) make chemical fat saturation inhomogeneous. Therefore, if pre-

gadolinium fat sat images are not obtained, you will not be able to determine whether bright spots on the post gad images are due to enhancement or due to inhomogeneous fat saturation. As opposed to the chest and superficial soft tissues, fat saturation is usually homogeneous in the abdomen and pelvis; hence, pre-gad fat sat images are not routinely necessary in abdomen and pelvis cases.

Dynamic imaging. When you give gadolinium, you should usually perform dynamic scanning with a rapid T1w sequence pre and post-gadolinium injection. As a general rule, 3d GE sequences are ideal for dynamic scanning. At UCSD, we use a 3d GE sequence called VIBE (volumetric interpolated breath-hold examination) to evaluate solid organs and we use a 3d GE sequence called MRA (magnetic resonance angiography) to evaluate vessels.

Delayed fat-saturated T1w images. We typically perform fat-saturated T1w 2d FLASH sequences after completion of the dynamic images. This usually occurs about 5 minutes after IV contrast administration, so we call these “delayed” images. As stated above, for chest and superficial soft tissue cases, make sure to get precontrast fat-saturated images to reliably evaluate contrast enhancement.

Sequences for Teslascan and Feridex. For Teslascan imaging, use in phase T1w 2d FLASH sequences. For Feridex imaging, use standard in and out of phase T1w 2d FLASH images and add a third 2d FLASH sequence with a TE of ~ 6.6 msec (“long TE”) to bring out some T2 weighting for increased sensitivity to Feridex. For now, we will also try a fourth 2d FLASH sequence with a TE of ~ 8.8 msec (“superlong TE”) to characterize FNH’s and assess Kupfer cell function.

Sequence to differentiate dephasing dues to flow from thrombus. Turbulent flow causes dephasing and signal loss, which can mimic a thrombus. Obviously differentiating thrombus from flow-related dephasing is important. One sequence that can be very helpful is **phase contrast**. Phase contrast is similar to color Doppler on ultrasound except it uses shades of gray rather than a color map. It gives you a map of flow velocity (in shades of gray) in a vessel. For example, fast forward flow is white, slow flow is gray, and reverse flow is black. To perform phase contrast, choose a direction of phase contrast (left-right, superior-inferior, or anterior-posterior) and a VENC (maximum velocity encoding). The VENC should be slightly higher than the maximum velocity you expect to see in a vessel. For example, you can’t tell if a dark signal in the portal vein is thrombus or turbulent flow. Choose a phase contrast direction of right-left and a VENC of 80 cm/sec. If you’re VENC is too small, you will get aliasing and flow direction will be mapped incorrectly. (For example, forward flow will be mapped as reverse flow).

Planes:

MR images can be obtained in virtually any imaging plane you can imagine. The most common imaging planes are standard axial, coronal, and sagittal planes, but oblique planes tailored to the patient's anatomy and pathology are often superior. As a general rule, choose the imaging planes that you think will best demonstrate a particular patient's anatomy and pathology. Coronal images are usually good for broad coverage and to visualize vessels and lymph nodes. Sagittal images are helpful for lymph nodes and to evaluate the uterus. Axial images are excellent for most solid organs. Angled axial or coronal images are ideal for specific body parts (cardiac chambers, aorta, uterus, ovaries, prostate, etc.). In the imaging protocols listed in the protocols handout, certain imaging planes are recommended for different indications. Remember that you can *and should* alter imaging planes as necessary to optimally tailor each study on an individual basis. This is especially critical when 3d volumetric sequences are not available. (With high-resolution volumetric 3d fast GE sequences such as VIBE, pixels are nearly isotropic and images can be reconstructed in various planes retrospectively).

Planes sometimes should be chosen to maximize coverage in the shortest amount of time. For example, in liver imaging: if the patient cannot breathhold, consider obtaining dynamic VIBE images in an oblique coronal plane so that the number of slices (or partitions) can be minimized. Ask your staff for help if you do not understand.

FOV:

As a general rule, we use large field of views (FOV's) for imaging the chest or abdomen and a small FOV for imaging specific body parts (heart, female pelvis, prostate, scrotum, etc.). Help the technician choose an ideal FOV on a case-by-case and sequence-by-sequence basis. Optimize the FOV to the region of interest. Be aware of tradeoffs, however, such as sacrifice in S/N and risk of wraparound (aliasing) with small FOV's. To eliminate or reduce wraparound, oversampling techniques, placement of sat bands, and appropriate choice of phase encoding direction can be helpful.

Other parameters:

Other important parameters include acquisition matrix and slice thickness. These will be listed in the specific imaging protocols. Keep in mind the following tradeoff: spatial resolution can be increased by increasing matrix size and decreasing slice thickness but at the cost of signal to noise and/or acquisition time.

6. Determining scan delay. At UCSD, we typically perform a timing run to determine optimal scan delay for dynamic gadolinium imaging. The timing run itself takes exactly one minute and tells us the "arrival time" (AT). Once we know the AT, we calculate the scan delay as follows:

$$\text{scan delay} = 1/2 \text{ injection time} + \text{AT} - 1/2 \text{ scan time}$$

If optimal timing is not critical and if the patient has no cardiovascular problems you can use a best guess approach. In normal-sized healthy adults, a scan delay of 0 seconds usually works for pulmonary arterial imaging, a scan delay of 12-15 seconds usually works for the thoracic aorta, and a scan delay of 15-20 seconds usually works for abdominal and pelvic imaging. However, there are many variables: anatomic location of region of interest, body habitus, cardiac output, size of heart and arteries, size and location of IV, bolus size, bolus rate, etc., etc. You can intuitively adjust your “best guess” by predicting how the various factors in any given patient will impact optimal scan delay. For example, based on the equation above, any variable that increases injection time (such as increasing the volume of contrast or decreasing the injection rate) or increases arrival time (such as using a smaller gauge IV or a more peripheral IV) will increase optimal scan delay while any variable that increases scan time (larger volume, greater number of slices or partitions) will decrease scan delay.

When in doubt, do a timing run.

Also, especially for vascular cases, err on the side of imaging a little too late rather than a little too early (ie, a scan delay that is 2-3 seconds too late is infinitely better than a scan delay that is 2-3 seconds too early).

How to Monitor a Case

Monitoring a case is just as important as protocolling a case. Sometimes it is even more important. As the case unfolds, scrutinize the images. Are you answering the clinical question? Is image quality adequate? Are imaging planes optimized to this particular patient’s anatomy and disease? Are technical artifacts present? Are there unsuspected abnormalities? Is patient able to cooperate with instructions? Are metal objects external to the patient degrading the image? If a metal object external to the patient causes artifact, remove it.

You may need to amend the original protocol to answer unsuspected questions or solve unforeseen technical obstacles. In particular, you may need to use tailored imaging planes optimized on a case-by-case basis.

In short, monitoring a case is your opportunity to be an artist. Create beautiful images! Answer the question! It’s fun!

MR Pulse Sequences: An Overview

As discussed briefly above, many imaging sequences are possible with multiple variations. These will be discussed in greater detail in a subsequent edition.

For now, just a few points:

1. In and out of phase imaging are very helpful in the liver (metabolic disease, focal hepatic lesions with steatosis such as many adenomas and a few HCC's), adrenal gland (adrenocortical adenoma vs. other adrenal mass), female pelvis (dermoid vs. hemorrhagic cyst), and occasionally in the kidney (AML vs. hemorrhagic cyst). Out of phase images are rarely helpful in other situations, so don't obtain them routinely. Even though they may require a very short time to acquire, they are a pain to sort through and look at on the Insight workstation and slow down case interpretation.
2. To detect ferridex effect in the liver, we perform GE sequences with TE = 2.2 (out of phase), 4.4 (in phase), and 6.6 (out of phase again, so-called "long TE" sequence). Note that as the TE increases from TE = 2.2 to TE = 6.6, the T2 weighting increases so the impact of Feridex increases. Tissues with functioning Kupffer cells progressively darken while those without functioning Kupffer cells do not. We exploit this to maximize detection of HCC in cirrhotic livers. We will also start using Feridex to characterize FNH and I have written a new protocol (Liver pre and post Feridex for FNH characterization) for this purpose. This protocol includes a "superlong TE" of 8.8. I'd like to try this for a while and see how it goes.
3. To stage retroperitoneal LN's we feel that sagittal and coronal T1w GE (2d FLASH) images are optimal. (Although SE sequences provide more signal to noise than GE sequences, the speed of the GE sequences more than makes up for this deficiency). Sagittal images should cover from the top of the kidneys to the perineum and from pelvic sidewall to pelvic sidewall. Coronal images should cover from the top of the kidneys to the iliac bifurcation (not to perineum!) and from 2 cm anterior to the anterior wall of the aorta to the mid VB of the most posterior VB within the scanning volume.
4. In chest cases, it is important to get T1w fat sat images *before and after* giving gad (fat saturation often inhomogeneous in the chest). In abdomen and pelvis cases, pre-gad fat sat images are not usually necessary (fat saturation usually homogeneous in the abdomen and pelvis).

Technical Tricks

Technical Tricks to Offset Respiratory Motion

1. Make sure patient is hyperventilated and coached for breathhold.
2. Use respiratory bellows so technologist can assess breathing real time.
3. Give O2 via NC.
4. Apply sat bands to eliminate the anterior body wall (or whatever body wall is causing the problem)
5. Reposition the patient to eliminate motion from the problematic body wall. For example, if the anterior body wall is moving place the patient prone.
6. Turn off the phase array opposite the side of interest.

7. Apply fat saturation to darken the fat, thereby lessening the impact of bright fat ghosts.
8. Switch from phase array to body coil (motion artifacts less severe with body coil)
9. Use breathheld rather than non-breathheld sequences
10. If breathheld sequences are still marred by motion, use ultrafast imaging: 2D turbo FLASH for T1 weighting, HASTE or FISP for T2 weighting.
11. If you must use non-breathheld sequences, one simple but time consuming method to offset respiratory motion is to “average out” the motion by increasing the number of excitations (from a NEX of 2, for example, to a NEX of 4 or 6). Other fancier techniques include respiratory compensation and respiratory triggering. These are not done at UCSD and will not be discussed.
12. Choose an imaging plane that maximizes coverage of the tissue or organ of interest in the shortest possible time. For example, choose an oblique coronal plane for liver imaging.
13. If contrast is more important than spatial resolution, obtain fewer slices of greater thickness. For example, obtain thirty 6-8 mm VIBE partitions rather than sixty 3-4 mm VIBE partitions.

Technical Tricks to Offset Susceptibility Artifacts at Surface-Air Interfaces

1. Turn pt into a cylinder: place Kaopectate, saline, or barium bags on the skin surface.

Technical Tricks to Offset Ghost Artifacts from Vessels

1. Switch phase and frequency encoding directions
2. Increase TR (this will increase the spacing between ghosts).
3. Apply saturation pulse upstream of the vessel causing the ghost to eliminate signal from that vessel.
4. Give gadolinium (ghost artifacts diminish because there is less difference between systolic and diastolic signal).
5. Apply GMN (gradient moment nulling) – this has a small theoretical benefit.

Technical Tricks to decrease blooming artifact

1. Increase Bandwidth (about 120 Hz/pixel)
2. Switch phase and frequency encoding directions
3. Change Plane of acquisition
4. Minimize TE
5. Increase echo train length (the greater the number of 180 refocusing pulses, the more correction there is for susceptibility errors, thus the less the susceptibility)

Techniques to Compensate for Cardiac Motion

To compensate for cardiac motion, EKG-triggering is necessary. This will be discussed in a later edition.

Techniques to Differentiate Flow Related Dephasing from Thrombus

Use phase contrast sequence (see earlier discussion under imaging sequences).

Fat Suppression Techniques

Some of the more frequently used fat suppression techniques include the following:

1. Frequency-selective saturation ("fat sat")
6. T1-dependent suppression (STIR)
7. Phase evolution (Dixon)
8. Selective water excitation (spatial spectral imaging)
9. Combined (SPIR)

These will be discussed in greater detail in a subsequent edition.

Coils

In general, use the following coils for the following indications

Indication	Coil
Abdomen and pelvis cases, pt < 20 cm diam	phase array
Abdomen and pelvis cases, pt > 20 cm diam	body coil
Abdomen and pelvis cases, pt unable to breathhold	body coil
Scrotum	surface coil
Prostate	anterior elements of phase array and rectal coil
Rectum	anterior elements of phase array and rectal coil
Brachial plexus	neurovascular coil
Superficial soft tissue mass	surface coil
Infants	knee coil
Breast cancer	Breast coil

These will be discussed in greater detail in a subsequent edition.

We encourage each of you, at the beginning of the rotation, to familiarize yourself with each of the coils. Ask one of the technologists to show you the coils so that you see what they look like and how they are used.

Key Equations and Tradeoffs

The following are some key equations that summarize tradeoffs inherent in manipulating imaging parameters. These are listed below. They will be discussed rather than merely listed in a later edition to enhance your conceptual understanding.

(1) Pixel size:

2DFT:

$$x = \text{FOV}_x / N_x$$

$$y = \text{FOV}_y / N_y$$

$$z = \text{THK}$$

3DFT:

$$x = \text{FOV}_x / N_x$$

$$y = \text{FOV}_y / N_y$$

$$z = \text{FOV}_z / N_z$$

(2) Voxel size = $x * y * z$

$$\text{2DFT: voxel} = \text{FOV}_x / N_x * \text{FOV}_y / N_y * \text{THK}$$

$$\text{2DFT: voxel} = \text{FOV}_x / N_x * \text{FOV}_y / N_y * \text{FOV}_z / N_z$$

(3) Measurements:

-- the number of measurements used in the Fourier reconstruction of a voxel

2DFT:

$$\text{measurements} = N_y * \text{NEX}$$

3DFT:

$$\text{measurements} = N_y * N_z * \text{NEX}$$

(4) Receiver bandwidth:

-- the range of frequencies that span a pixel in the frequency encoding direction;
note that this is **not** the same as transmit bandwidth

$$\text{BW} = N_x / \text{sampling time}$$

(5) $\text{SNR} \propto \text{voxel size} * \text{square root (measurements)} / \text{square root (receiver BW)}$

(5a) SNR \propto voxel size

(5b) SNR \propto 1/Resolution

(5c) SNR \propto square root (measurements)

(5d) SNR \propto square root (N_y) * square root (NEX) 2DFT

(5e) SNR \propto square root (N_y) * square root (N_z) * square root (NEX) 3DFT

(5f) SNR \propto 1/square root (receiver BW)

(6) Time =

$$2DFT: \text{time} = \frac{TR * N_y * NEX}{ET}$$

$$3DFT: \text{time} = \frac{TR * N_y * N_z * NEX}{ET}$$

(7) Comparison of 3DFT and 2DFT (all other factors equal)

$$SNR_{3DFT} = SNR_{2DFT} * \text{square root } (N_z)$$

$$T_{3DFT} = T_{2DFT} * N_z$$

(8) Image degradation \propto Total scan time

NEX = number of excitations or acquisitions or signal averages

FOV = field of view

N_x = number of frequency encoding steps in x-direction

N_y = number of phase encoding steps in y-direction

N_z = number of phase encoding steps in z-direction (3DFT)

-- defined as the number of partitions in z-direction

ST = slice thickness

MR Contrast Agents: An Overview

In the United States, currently available MR contrast agents include vascular/extracellular agents (Gd chelated to DTPA or to DTPA derivatives), RES agents (superparamagnetic iron-based particles), and hepatocellular/biliary agents (manganese chelated to DPDP). Combined extracellular and hepatocellular agents are approved in Europe (Gd chelated to BOPTA) or are in clinical trials (Gd chelated to EOB-DPTA) but are not yet available in the U.S.

Unfortunately, insurance companies generally pay for a single contrast agent using the standard dose. If you decide to use two contrast agents to achieve a synergistic effect or if you decide to double the dose of contrast to increase sensitivity to enhancement, insurance companies will probably not reimburse you for the extra cost.

1. Vascular/Extracellular Agents

Gadolinium can be chelated to large ligands such as DTPA to form very effective, nonspecific vascular/extracellular MR contrast agent agents. These gadolinium (Gd)-based compounds are the most widely used MR contrast agents worldwide. Four commercial formulations are currently available, in which Gd is complexed to DTPA (Magnevist by Berlex) or to one of three DTPA derivatives: DOTA (Dotarem by Guerbet), DTPA-BMA (Omniscan by Nycomed), and HP-D03A (ProHance by Bracco). The four formulations confer minor differences in osmolality and chemical stability, but these differences are probably not clinically important.

These Gd chelates have similar pharmacokinetics as iodinated contrast. They enter perfused tissues and freely redistribute from the vascular to the interstitial space, while being excreted by the kidneys. These chelates do not pass cell membranes and have negligible hepatocellular uptake and biliary excretion.

A paramagnetic heavy metal with seven unpaired electrons, Gd shortens the T1 relaxation of adjacent water hydrogens, making tissues more intense on T1w images. (For Gd to work it must be exposed to the “hydration layer”. If Gd is placed in a chemical “cage” that shields the Gd from the hydration layer, the Gd will cause no T1 shortening unless the cage is broken). Because MR is so exquisitely sensitive to Gd, subtle areas of contrast material accumulation can be detected on T1w MR images that would be missed on CT using iodinated contrast. Gd also shortens T2 relaxation, and in very high concentration, the T2-shortening effect predominates, causing tissues to lose signal. The T2 shortening effect can be an issue in renal and bladder imaging as Gd becomes more and more concentrated in the urine, but this is not an issue in liver imaging because the Gd concentration is sufficiently low that the T1-shortening effect is paramount.

Dosing. Gd is given as a bolus intravenous injection at a rate of 2-3 mL/sec. (At UCSD, we use a power injector for essentially all adult cases). Recommended dose is 0.1 mM/kg. For Gd-DTPA, 1 mM = 2 ml (0.5 mM/ml). Therefore, this corresponds to 0.2

ml/kg body weight (roughly 1 ml/10 lbs body weight). For practical reasons, we give almost all adults the same 20 ml dose, even patients considerably lighter than 200 lbs.

Again, do double dose for heavy patients or for vascular cases.

Adverse reactions. Gd-based compounds are generally very safe. Moderate adverse effects (bronchospasm, laryngospasm, facial edema, arrhythmias, widespread urticaria) occur in 1:5000 adults. Severe anaphylactoid reactions including death occur in 1:400,000. Atopic individuals may be at slightly higher risk. Infants and children seem to tolerate Gd well and the risk of an adverse reaction is probably similar in the pediatric and adult populations.

Pregnant women. Gd-compounds are class C drugs, which means that they have been shown to cause adverse effects in fetuses in animal models (skeletal malformations were seen in rabbits given 2x the human dose for 13 days; malformations were not seen in rats). The safety of Gd has not been confirmed in human fetuses and Gd should not be administered routinely in pregnant women unless the benefits clearly outweigh the risks.

Nursing mothers. It is unknown if Gd is excreted in human milk. Even if it is excreted, the risk to the baby is probably very small as Gd is generally well tolerated in infants and children. To be on the cautious side, if a breast-feeding woman receives a dose of Gd, she should probably withhold breastfeeding for 24-48 hours.

Cost. Gd is the least expensive of the available contrast agents, costing ~ \$60/20 cc vial.

Clinical use: Gd chelates are the workhorse in MR imaging. They are used to visualize vessels and to detect lesions. The enhancement pattern of a lesion is often very helpful in characterizing the lesion as benign or malignant and sometimes (eg, hemangioma) is sufficient to render a specific diagnosis.

2. Reticuloendothelial system (RES) agents

As opposed to the nonspecific vascular/extracellular Gd-based compounds discussed above which redistribute within the extracellular space of the liver but have no hepatocellular uptake, contrast agents that target the RES system and are entrapped by RES macrophages have been developed as liver-specific agents. The most widely available RES agents are ferumoxides, small (30-150 nm) iron-based particles coated with polysaccharides such as dextran and administered as a dark brown injectable colloid. These iron-based particles are commercially available under the brand name Feridex (Berlex) in the US and under the brand name Endorem (Guerbet) in Europe. The particles are phagocytosed by macrophages throughout the RES system, including liver, spleen, lymph nodes, bone marrow, and lung. Because of their size, however, the particles are preferentially entrapped by the Kupffer cells lining the liver sinusoids, and over 70% of the injected dose accumulates within the normal liver, the rest accumulating in other RES components. In cirrhotic patients, the particles accumulate within regenerating nodules but not within areas of scarring and fibrosis. Cysts and most

hepatic neoplasms (whether benign or malignant, primary or secondary) lack or are deficient in Kupffer cells, and the particles tend not to accumulate within focal lesions, although minor entrapment may occur, probably because of leakage through tumor capillaries. On the other hand, some hepatic neoplasms – eg, some dysplastic nodules, well-differentiated HCC, FNH, and rarely adenoma – may contain Kupffer cells and will accumulate the particles to a variable but potentially significant extent.

The iron particles are superparamagnetic and cause significant local magnetic field inhomogeneity. Nearby protons are dephased, resulting in T2 and T2* shortening. T1 shortening also occurs, but unlike Gd chelates, the T2 and T2* shortening predominates. As a result, tissues that accumulate the iron particles show reduced signal intensity, particularly on T2w and T2*w images, and to a lesser extent on T1w images.

Dosing. Feridex must be infused very slowly for at least 30 minutes and preferably 60 minutes through an intravenous line with a 5 micron filter at a rate of 2-4 ml/sec. The recommended dose is .05ml/kg body weight (~1ml/40 lbs) diluted in 100 ml of D5W. Because it takes time for the particles to be phagocytosed and to accumulate, imaging should be done approximately 45-60 minutes after termination of the infusion but can be delayed up to 3.5 hours if needed for logistical reasons. The iron particles remain visible in RES tissues (liver, spleen, marrow, etc.) for several days but are gradually destroyed by lysosomes within the RES cells. The liberated iron then enters the normal iron metabolic pathways. The total amount of iron administered in a single Ferridex infusion corresponds to approximately 4% of the total blood pool of iron and is considerably less than total body stores. This should not be a problem except in patients with iron overload states, in whom the use of Feridex is relatively contraindicated.

Adverse reactions. Unlike Gd chelates, which are very safe, adverse effects are common with iron oxides. Anaphylactoid and allergic adverse events (generalized urticaria, respiratory symptoms, and hypotension) requiring treatment occur in ~1:200. One vexing problem associated with iron oxides is acute severe back, leg, or groin pain. Approximately 3% of patients will experience acute pain of sufficient severity to discontinue or interrupt the infusion. Unfortunately, the rate of adverse reactions is higher in patients with cirrhosis, precisely the population that may benefit the most from Feridex administration. Very slow infusion of the drug helps to reduce this risk. At UCSD, we try to infuse the agent over a full hour.

Pregnant women. Feridex is a class C drug – it is teratogenic in lab animals – and should not be administered to pregnant women unless the benefits far outweigh the risks.

Nursing mothers. It is unknown if Feridex is excreted in human milk. Because the safety of Feridex in infants is also unknown, the drug should not be given to nursing mothers unless the potential benefit clearly outweighs the risk.

Cost. Feridex is relatively expensive at nearly \$150 per 10mL vial. We almost never use Feridex by itself but rather as a supplement to Gd, so third-party insurers do not cover the cost of the agent.

Clinical use. At UCSD we usually reserve Feridex for detection of HCC in cirrhotic livers or high risk patients. A potential application of Feridex is to differentiate between adenoma (theoretically should have no or minimal uptake) and FNH (may have significant uptake). We are about to start using Feridex for this purpose at UCSD and a new protocol has been drafted (Liver pre and post Feridex for FNH). This protocol incorporates a “superlong TE” 2D FLASH (TE=8.8).

3. Hepatocellular/biliary agents

Contrast agents that target hepatocytes rather than RES cells have also been developed as liver-specific agents. The most widely available is mangafodipir, a chelate of manganese and DPDP, Mn-DPDP, which is commercially available in the US under the brand name Teslascan (Nycomed). Manganese has five unpaired electrons and is a powerful T1 relaxation agent, rendering tissues hyperintense on T1w images. There are two pathways for Mn accumulation in the liver after IV injection of Mn-DPDP. First, the Mn-DPDP chelate has a similar chemical structure as vitamin B6 and, after intravenous injection, the entire chelate is taken up by hepatocytes via the vitamin B6 metabolic pathway. Second, within the blood, endogenous zinc displaces some of the manganese to form Zn-DPDP chelates, liberating Mn⁺² ions. The free Mn⁺² ions circulate in the blood and are taken up by hepatocytes in the liver via nonspecific mechanisms (they are also taken up by the pancreas, adrenal gland, renal cortex, and myocardium, resulting in enhancement of these structures as well). Hepatic parenchymal enhancement gradually increases as Mn, in free ionic form and in chelated form, accumulates within hepatocytes. Enhancement peaks at approximately 15 minutes, and persists for several hours, allowing an imaging window of up to 4 hours. Within about 5 minutes of injection, normal bile ducts also begin to enhance as the Mn⁺² ion is excreted into the biliary system, and the enhancement progressively increases over the next several minutes. Obviously, it takes longer to fill and enhance obstructed bile ducts.

Dosing. Teslascan is administered intravenously at a dose of 0.1 mL/kg (~0.5 mL/10 lbs) relatively slowly over one minute, with maximum dose of 15 mL. For lesion characterization, we generally image the liver at 3, 5, and 10 minutes after Teslascan injection. For biliary cases, we image at 5, 10, 15, 20 minutes, ± 30 minutes, ± 60 minutes.

Adverse reactions. The drug appears to be very safe. The most common adverse effects are headache (5%), nausea (11%), and vomiting (3%). Severe anaphylactoid events have not been reported to our knowledge.

Pregnant women. Teslascan is a class C drug (causing both toxic and teratogenic effects in animal fetuses) and should not be given to pregnant women unless the benefit clearly outweighs the risk.

Nursing mothers. Manganese is excreted in human milk, but the extent to which manganese or DPDP is excreted after Teslascan injection is unknown. Nursing mothers should probably temporarily discontinue breast feeding if they receive a dose of Teslascan.

Cost. At nearly \$85/10 cc vial, Teslascan is intermediate in price between Gd-DTPA and Feridex.

Clinical use. After IV injection, hepatocyte containing masses (HCC, FNA, and adenoma) generally enhance while non-hepatocyte-containing masses such as metastases do not. Thus, the major utility of Teslascan is in differentiating lesions of hepatocellular from those of non-hepatocellular origin. In clinical trials, 75% of lesions of hepatocellular origin enhance while only 25% of lesions of non-hepatocellular origin enhance. (The mechanism of enhancement of the non-hepatocellular lesions is unclear but may be related to non-specific uptake of free Mn⁺² or to leakage of the chelate through tumor capillaries). Although the presence of enhancement after Teslascan injection is useful in establishing a lesion as being hepatocellular in origin, the pattern of enhancement is not reliable in differentiating benign from malignant hepatocellular lesions as there is considerable overlap in enhancement patterns.

Because it is excreted into and enhances the biliary system, Teslascan is also helpful in complex biliary cases, especially when conventional MRCP is non-diagnostic. Delayed imaging is usually necessary to fill obstructed bile ducts.

4. Combined vascular/extracellular and hepatocellular agents.

Gd-BOPTA (marketed by Bracco under the brand name MultiHance) is available in Europe as a combined extracellular and hepatocellular agent. The ligand, BOPTA, is similar to DTPA. Hence, Gd-BOPTA behaves like an extracellular agent during its first pass through the liver, and can be imaged dynamically after bolus injection to assess tumor vascularity. However, because of the addition of a benzene ring and a short carbon chain along one of its arms, BOPTA is also taken up by hepatocytes via membrane-bound transporters and subsequently excreted into the bile. Delayed imaging therefore can be performed to characterize lesions as being hepatocellular or non-hepatocellular in origin. A similar drug, Gd-EOB-DTPA (produced by Schering) is in clinical trials. Neither is currently available in the US.

Abdomen

Liver

Advances in MR imaging software have allowed for fast imaging techniques that provide T1 and T2-weighted sequences. The fast T2 technique is the fast or turbo spin echo with 25 to 32 echo-trains to allow for breath-holding. The HASTE (**Half-Fourier single-shot turbo spin echo**) on Siemens or the single shot fast spine echo (SSFSE) T2 on GE decrease the limitations of MRI due to motion and provide the opportunity for MR cholangiopancreatography (MRCP) and MR Urography. Although non-contrast T1w and T2w MR imaging provides better lesion detection and characterization than non-contrast CT, contrast media is required for adequate liver imaging using either modality. Overall, MR is comparable to CT for lesion detection in most settings and appears to be superior to CT in patients with cirrhosis in whom liver perfusion is altered or impaired. MR is essential in patients unable to receive iodinated contrast.

Equally important to detection is the ability to characterize lesions as benign or malignant. Although faster CT scanning has improved the ability to distinguish hemangiomas from metastases, MR is superior in its ability to assess several lesions located at multiple levels. The reason for this is that the entire imaging volume is acquired simultaneously with MR while it is acquired sequentially with CT. The faster multi-array detector CT scanners have partially overcome this disadvantage. Noncontrast MRI allows the differentiation of hemangiomas from hypovascular lesions (colon metastases) but not from hypervascular lesions (endocrine tumors) by their appearance alone (bright on T2, free of internal echoes, and sharply demarcated from liver). IV contrast adds to MR characterization because of the unique enhancement pattern of hemangiomas (peripheral discontinuous puddling, gradual and near complete filling, and longer dwell time than liver). Therefore, a well-demarcated lesion that is very bright on T2, has no internal architecture, shows puddling of contrast at the lesion's edge and progressively fills in is a hemangioma. Thus, MR is generally superior to CT for lesion characterization (except in the rare lesion in which detection of calcification is important, in which case CT is superior).

As described earlier on the section on MR contrast agents, iron oxide (Feridex, Berlex Laboratories), an RE agent, and Mn-DPDP (Teslascan, Nycomed Amersham), a hepatocyte specific agent, have been approved for lesion detection and some centers use these agents routinely for detection of liver metastases. In addition to improving lesion detection, Mn-DPDP is capable of differentiating hepatocellular lesions (adenoma, FNH, HC carcinoma) from metastatic lesions as well as assessing liver excretion and the hepatobiliary tree. We use Feridex almost exclusively in cirrhotic or high-risk patients to improve detection of HCC. By decreasing background liver signal, Feridex makes HCC lesions stand out as hyperintense masses relative to liver on pre-gad images, particularly on relatively T2w images (eg, the "long TE" GE sequence and the modified TSE T2w sequence). Reduction of background liver signal also increases the contrast-to-noise ratio of lesions relative to liver following Gd administration. In addition to improving HCC detection in high-risk patients, we are about to start using

Feridex to differentiate FNH from other hepatocellular lesions. Currently, we use Teslascan in select patients in whom the recognition of a hepatocyte-containing vs. a non-hepatocyte-containing lesion is critical.

Biliary tree and pancreatic duct: MRCP

The biliary tree and pancreatic duct are well evaluated with MRCP. The key sequence in MRCP is HASTE. HASTE sequences can be obtained with an effective TE ranging from 80 to 1000. The optimal effective TE for MRCP is controversial and different institutions have different preferences. In general, as the effective TE is increased, the T2 weighting of the image increases, which enhances the conspicuity of the bile and pancreatic ducts, but signal from background soft tissues diminishes, reducing anatomic orientation. At UCSD, we prefer an effective TE of 180 to 300 msec for the thin HASTE images but a TE > 1000 msec for the thick slabs. The thick slabs are obtained with fat saturation to further reduce background signal. In complex biliary cases, we sometimes give Teslascan to assess hepatocyte function and excretion, opacify bile ducts and delineate biliary anatomy, and confirm potential biliary stenoses or bile leaks.

Spleen

The spleen is a frequent site of metastatic disease in melanoma and in hematological malignancies such as lymphoma. Accurate detection of splenic Hodgkin disease is necessary to obviate the need for surgical resection for staging. (Staging of NHL for splenic involvement is not as critical since NHL is usually considered and treated as a systemic disease even if imaging suggests that it is anatomically confined to one organ). If liver imaging for lesion detection is challenging then splenic imaging is nearly impossible. The spleen is not only smaller than liver, it displays pseudolesions on dynamic CT and MR due to the inhomogeneous wash-in rate of contrast media. To add to the difficulty in imaging the spleen, MR relaxation parameters of splenic parenchymal, unlike those of liver parenchyma, are similar to those of splenic lesions. Focal masses and infiltrative disease such as lymphoma generally display poor contrast with the normal spleen since both have similar T1 and T2 relaxation times. Furthermore, unlike the liver, which has a dual blood supply, the spleen has a single blood supply. Hence, normal splenic tissue and pathologic lesions are both fed by the splenic artery and enhance simultaneously. When necrotic, cystic, or hemorrhagic, lesions can be well demonstrated, however. RE-contrast agents, such as Feridex have had mixed results.

Pancreas

Pancreatic tissue is as bright or slightly darker than liver on T1 weighted images and is slightly brighter than liver on T2 weighted images. The common bile duct and pancreatic duct are routinely seen on the HASTE and TSE T2-weighted sequences allowing their assessment in normal, anomalous, or obstructive conditions.

Although pancreatic tumors can assume a slightly darker signal on T1 weighted images, and slightly brighter signal on T2-weighted images than normal pancreatic

tissue, the classic pancreatic cancer is typically isointense with pancreas. Marked hyperintensity within a solid mass on T2-weighted images suggests hypervascularity (as in an islet cell tumor), tumor necrosis, and/or hemorrhage. Imaging of the pancreas requires the use of IV contrast and is best done with dynamic T1w fat-suppressed breath-hold sequences. As with CT, most pancreatic cancers are hypovascular relative to the normal pancreas but may have greater enhancement during the arterial phase. The sensitivity and specificity of the new sequences in staging pancreatic cancer is not yet known but appears to be at least comparable to CT. The new liver agent Teslascan has been shown to accumulate in pancreatic tissue to produce specific pancreatic enhancement. The utility of such an agent in neoplastic and inflammatory diseases of the pancreas are not yet well defined. We have used Teslascan to highlight the pancreas to ease its visualization in post-operative patients.

Kidneys

The anatomic structures of the normal kidney are clearly defined on MRI. The fat in the renal hilum has high intensity signal, vessels have low signal intensity "flow void", and the pelvis has intermediate intensity like water. Distinction between the renal cortex and medulla is evident where the medulla assumes a darker signal on T1-weighted images (the medulla has greater "water content" than the cortex and hence is darker on T1w images). Urine in the renal pelvis is of lower signal than renal tissue on T1 and assumes a brighter signal on T2 weighted images. The breath-hold T2 weighted sequences can demonstrate the urine-filled structures (kidneys, ureters, and bladder) in a way analogous to the biliary tree providing images comparable to an IVU. Some studies assessing the value of HASTE-type imaging following Lasix administration have suggested that exquisite anatomic detail can be achieved.

IV contrast is required to optimally image the kidneys with MR. Although Gd chelates are handled by the kidney in an identical fashion to iodinated agents, there are a few differences in their impact on renal and urinary enhancement. Unlike CT, the dose response of MR signal to Gd is non-linear. It first increases at low concentrations because of T1 shortening and then decreases at higher concentrations as T2-shortening predominates. Hence, the collecting systems, ureters, and bladder first brighten and then darken as the gadolinium concentration within the urine increases. The second difference is that MR is very sensitive to small quantities of Gd. Hence, renal cancers tend to enhance to a greater degree on MR than on CT, and the contrast between kidneys and cancer on MR is not as great as it is on CT. For the same reason, however, MR is more sensitive than CT for detecting small foci of enhancement in cyst walls is therefore superior to CT for characterization of complex cysts and small masses. Otherwise, With the advent of newer faster CT scanners, MR imaging and CT are generally comparable.

MR imaging with IV contrast is best done in a dynamic fashion. Fast gradient echo series covering the entire kidney in the best plane demonstrating the lesion and kidney interface are obtained pre- and then during the arterial, venous, and delayed phases of contrast. We routinely evaluate the kidneys on dynamic scanning using the

3D-VIBE technique, as it provides appropriate spatial and contrast resolution and also allows for multiplanar reformation to assess vessels and three dimensional relationships.

Adrenal Glands

The right adrenal gland, truly suprarenal, is located superior to the upper pole of kidney, lateral to the right crux, medial to the liver and posterior to the inferior vena cava. In contradistinction, the left adrenal gland is not truly suprarenal; it is located anterior and medial to the upper pole of the kidney and lateral and posterior to the aorta. Its position relative to the left crux and left splenic and renal arteries is variable leading to potential confusion with these structures. Further, unlike the right adrenal, which is distant from bowel structures, a gastric fundal diverticulum can present posterior to the splenic artery on axial scans and mimic an adrenal mass.

The approach to the patient with potential adrenal pathology is key to minimizing the imaging studies necessary to reach a diagnosis. For patients with functional adrenal lesions in whom the goal of imaging is lesion localization, CT, with its superior spatial and time resolution, is the better study, as functioning lesions tend to be small and are infrequently bilateral. MR is useful if disease is bilateral or if extra-adrenal pheochromocytoma is sought.

The main role of MR is in characterizing adrenal lesions typically encountered in cancer patients on CT scans. Because of the superior contrast resolution of MR, it has been able to distinguish metastatic disease from nonfunctioning adenomas. This is accomplished by assessing the lesion's signal on T2-weighted images relative to liver and fat. Metastases and pheochromocytomas are brighter than liver and as bright or brighter than fat. Adenomas are darker than fat and as dark or darker than liver. These characteristics are true in 75-80% of cases when imaged with the standard spin-echo techniques. The crossover of signal occurs when adenomas are bright on T2.

Adenomas, even when non-functioning, typically contain a large amount of cholesterol or cholesterol-like molecules, which are rich in $-CH_2-$ protons. Because these $-CH_2-$ protons resonate at a frequency 220 Hz below that of water protons at 1.5 Tesla, it is possible with gradient echoes to acquire images in which the two proton populations are in-phase (they add) or opposed-phase (they subtract). On opposed-phase images, voxels that contain a mixture of fat and water, such as those at organ margins or when lesions are a mixture of fat and water, lose signal relative to voxels containing only fat or only water protons. Therefore, when adrenal masses are compared to spleen on in-phase and opposed-phase images, adenomas become darker while metastases do not change. Although the gradient echo in-phase and opposed-phase technique is highly *specific* for adenoma, it is not *sensitive* for adenoma. In other words, an adrenal lesion that darkens on opposed-phase images can be safely considered a benign adenoma. An adrenal lesion that does not darken may or may not be a benign adenoma.

Note that non-enhanced CT (NECT) evaluation of adrenal adenomas works on the same principle: an adrenal lesion that is lower than 10 HU on NECT is inferred to

have abundant intralesional fat (cholesterol) and is therefore a benign adenoma. An adrenal lesion that is greater than 10 HU on NECT may or may not be a benign adenoma.

Because in- and opposed-phase MR and NECT work on the same principle, an indeterminate lesion on MR will be indeterminate on NECT and vice versa. Therefore, don't do an MR to characterize an adrenal mass if the patient has already had an indeterminate NECT. Suggest a washout contrast-enhanced CT study instead (look for > 50% washout at 15 minutes).

Arteries

With the advent of rapid breath-hold imaging and the ability to perform rapid 3D acquisition in a single breath-hold, MR angiography has become a simple procedure. This technique relies upon the use of IV contrast and requires proper timing to achieve optimal results. Because image contrast is dominated by the data acquired during the middle 1/3 of the acquisition, a delay-time has to be incorporated to time the peak bolus effect in the region of interest with the middle of acquisition. The timing run consists of 40 axial slices over the region of interest acquired one image per second that begin with the beginning of injection of 2ml Gd followed by 15-20 ml of saline given at 2 ml/sec for solid organ imaging and 3ml/sec for MRA. The image number with greatest enhancement indicates the arrival time (AT) from start of injection.

As discussed earlier, you can calculate the optimal scan delay (the delay time between the beginning of injection and beginning of scanning) as:

Scan delay = arrival time – 0.5 acquisition time + 0.5 injection time.

Injection time is typically 10 seconds for solid organ imaging (20 ml gad @ 2 ml/sec) and 13 seconds for MRA (40ml Gd @ 3 ml/sec). Acquisition time is typically about 20-25 seconds. To ensure adequate breathhold during the acquisition, the patient is given breathing instructions and hyperventilated prior to scanning. Breathing instructions typically commence about 9 seconds before beginning of scanning and about 1 – 4 seconds after beginning of contrast injection.

Veins

MR venography can also be performed with dynamic gadolinium-enhanced 3d GE techniques. The same MRA protocol can be used as above but more delayed images coinciding with peak venous enhancement are obviously critical to evaluate veins. Delayed fat saturation images are particularly useful to detect thrombi.

If the patient has no venous access, then 2D time of flight (TOF) sequences such as true FISP can be used to evaluate veins as these do not require gadolinium. (3D TOF may be helpful in CNS imaging but is usually problematic in body imaging because

of long acquisition times). At UCSD, we almost never use 2D or 3D phase contrast (PC) sequences.

Lymphnodes

The goal in MR imaging for the detection of lymph nodes is to demonstrate anatomy in the most optimal plane and to maximize contrast between nodes and surrounding tissues. Fortunately, since the most common tissue surrounding nodes is fat and since fat has a very short T1 while nodes have longer T1, T1-weighted sequences are ideal for node detection. T2-weighted sequences are poor for nodal detection but are excellent for characterization to allow the distinction of nodes from muscles, vessels, or scars.

The optimal imaging plane is that with which the observer is most skilled. However, gaining expertise in the sagittal and coronal display of anatomic relationship confers a significant advantage. For instance, the entire retroperitoneum can be viewed and adequately assessed with one T1-weighted sequence, in either the sagittal or coronal plane. The pelvis can be fully evaluated with a sagittal T1-weighted sequence. In fact, we believe that sagittal views of the pelvic sidewall are easier to interpret than axial scans, particular when nodes are small. In the retroperitoneum and pelvis, vessels and most of their tributaries travel in the sagittal or coronal plane. Hence on sagittal and coronal images, nodes (circles) are easily differentiated from vessels (cylinders). In our protocols, we utilize the sagittal and coronal T1-weighted sequences as our only methods to stage nodal enlargement in cancer patients.

Similar to CT, nodal size is relied upon to determine the likelihood for malignant involvement. But nodal size is nonspecific and MR cannot reliably distinguish benign from malignant lymphnodes, although there are a few helpful features. For example, lymph nodes that are dark on T2weighted sequences (as dark as muscle) are unlikely to be malignant. On the other hand, lymph nodes with central necrosis (needs IV contrast) are more likely to be malignant.

From our experience and that of others, MR is comparable to CT in the detection of nodal enlargement in most parts of the body. There are, however, anatomic regions that are best imaged with sagittal or coronal projections (eg, the cervico-thoracic junction, supraclavicular region, lung apex, AP window, and pelvis). In these regions, and especially if the patient is not able to receive iodinated agents, MR is a better tool than CT.

On the horizon are specialized contrast media developed uniquely to enhance lymphnodes providing MR with indirect lymphographic techniques. One utilizes ultrasmall iron particles that can escape the capillaries and be carried through the lymph to enhance the normal part of the node. Another enhances lymphnodes following the administration of contrast material directly in the subcutaneous space along the path of drainage. These techniques are still under development.

Female Pelvis

MR imaging displays the normal and abnormal female pelvic anatomy with exquisite detail and high contrast in any plane. It is the imaging modality of choice to demonstrate congenital anomalies of the uterus. MRI is also sensitive to changes in size and signal of the uterus and ovaries as hormonal influences vary with the menstrual cycle as well as with birth control pills and menopause. Sonography remains the first line of defense because of its versatility, accessibility, portability, and cost. However, should the sonogram be equivocal, inconclusive, or does not answer the clinical question, MR imaging should be done. We and others have shown that the increased specificity afforded with MR adds important clinical data in 75% of cases regardless of whether sonography was inconclusive or of sufficient quality.

Patient preparation. If fast imaging is not available on the scanner, patients should be NPO at least 3 hours to minimize urine production during the exam period and the alteration of pelvic anatomy. Patients are asked to empty their bladder just prior to scanning and 60ml of Surgilube is injected in the vagina. They are instructed not to squeeze the gel out. This is accomplished by cutting a Yankauer Suction Tube at the middle of the bulge and attaching to it a 60ml syringe with NG tip filled with Surgilube (a full tube). The patient inserts the catheter in the vagina and the technologist injects the Surgilube. Surgilube, like water in signal, is useful to define the cervix and vaginal fornices.

Technique. To assess uterine anomalies, use the Female pelvis, uterine anomaly protocol. To assess adnexal masses or fibroids, use the Female pelvis, limited protocol. To assess or stage ovarian, uterine, or cervical cancer, use the Female pelvis, complete with abdomen protocol.

To maximize throughput and minimize charge, the Female pelvis, limited protocol is designed to take only 15 to 20 minutes and utilizes rapid breath-held sequences exclusively. More exquisite anatomic detail could be obtained with non-breathheld sequences but this would increase imaging time without providing additional clinical information in this patient population.

On the other hand, many women with gynecologic malignancy have distorted anatomy related to tumor invasion or to prior surgery and/or radiation. In these women, it is crucial to differentiate post-operative/radiation change from neoplastic tissue, and non-breathheld pre-contrast sequences of the pelvis are usually necessary to achieve sufficient anatomic detail for confident diagnosis. After gadolinium administration, dynamic imaging is performed using a 3D fast GE sequence (eg, VIBE). Delayed fat saturated images are then obtained using a rapid breathhold T1w GE sequence. A breathhold sequence suffices for delayed imaging since the purpose of the delayed scan is to identify areas of residual enhancement rather than to delineate anatomic detail.

One more point: notice that in the Female pelvis, complete protocol, precontrast images of the abdomen are *not* obtained (except for the coronal and sagittal T1w images used to stage retroperitoneal lymph nodes and coronal HASTE images used to

detect hydronephrosis). The abdomen is evaluated for metastatic disease only on delayed fat-sat T1's (axial and coronal planes). This makes sense from a biological point of view because the delayed fats should be adequate for detection of serosal and peritoneal metastases. The pre-test probability of parenchymal as opposed to serosal solid organ metastases is vanishingly small so precontrast images through solid abdominal organs is not necessary.

Discussion

Uterine Anomalies

Uterine anomalies are usually duplications. A septate uterus is easily distinguished from a double-horned uterus (bicornuate – 2 horns with single cervix; didelphys – 2 horns with 2 cervices) on MR imaging. The distinction is best accomplished on a true coronal scan through the uterus by assessing the outer curvature of the uterine fundus. A septate uterus has a normal smooth curve (convex outward), while a double-horned uterus has an indented or concave outer surface. Further, true axial T2 weighted images show the septate uterus with one complete junctional zone and a dark central band (the septum) while a double-horned uterus has two complete junctional zones separated by myometrial signal. It is therefore imperative to obtain true axial and coronal T2 scans of the uterus. Not uncommonly, a septate uterus has myometrial tissue at the fundal aspect of the septum. The myometrial tissue extends inferiorly to a variable degree before the septum becomes purely fibrous. Somewhat ironically, although a septate uterus represents a later anomaly in embryologic development than a double-horned uterus, a septate uterus confers greater infertility risk. One somewhat simplistic but plausible explanation is that the fibrous portion of the uterine septum acts as an unfavorable site for implantation.

Vaginal duplications are more difficult to diagnose on MR (but should be easier on physical exam). Surgilube can be of benefit.

Fibroids

Fibroids are in general easily detected and defined on MR. The great majority of fibroids are intermediate on T1 and dark on T2 owing to their fibrous and muscular content, which decreases their signal on T2-weighted images. Given that ovarian masses are typically bright on T2, MR is of benefit in differentiating adnexal masses of ovarian origin from subserosal or pedunculated fibroids. In addition to their ability to masquerade as adnexal lesions, fibroids can also cause bleeding and infertility. With the availability of medical, embolic, or targeted surgical therapy, the delineation of number, size, and precise location of fibroids has become important. For this purpose, MRI is the best imaging modality. Such fibroids are typically submucosal in location. At UCSD, we diagnose submucosal fibroids as those that efface the transition zone and cause mass effect on the endometrium. The differential diagnosis for a mass that effaces the junctional zone and is dark on T2 is an adenomyoma (a focal masslike form of adenomyosis) but, unlike submucosal fibroids, adenomyomas do not usually distort the endometrium.

Although the majority of fibroids are intermediate on T1 and dark on T2, these tumors can have more variegated signal characteristics owing to their diverse histology and variable degrees of degeneration. For example, many fibroids assume signals of higher intensity than normal myometrium on T1-weighted sequences and may be as bright or even brighter than myometrium on T2-weighted sequences. Liquid degeneration of fibroids is also common. The area of liquefaction, which is usually central in location, assumes signals similar to those of water. Sometimes the liquefaction is peripheral and eccentric rather than central. Fibroids that have undergone liquid degeneration invariably have regions of typical fibroid signal (dark on T2).

Statistically, a highly heterogeneous myometrial mass is probably a degenerated fibroid, but if the woman is postmenopausal then a leiomyosarcoma should be suspected.

If an adnexal mass assumes signal characteristics similar to a fibroid (intermediate on T1 and dark on T2), it is important to assess the site of origin. Such a mass could be a fibrous tumor of the ovary or a solid endometriosis deposit.

Adenomyosis

This disease is caused by the presence of ectopic endometrial tissue within the myometrium, and causes abnormal bleeding, heavy periods, and at times pain. Because it is the basalis endometrium, which is insensitive to hormones, that invades the myometrium, this disease is not treatable medically and requires hysterectomy for control. The invading endometrial tissue causes fibrous and smooth muscle hyperplasia, which, in turn, thickens the junctional zone. Although diffuse involvement can cause diffuse symmetrical thickening of the junctional zone to become greater than 1/3 to 1/2 of the myometrial thickness, focal involvement causes asymmetric fusiform thickening without mass effect on the endometrial signal. However, at times such focal changes can be difficult to distinguish from submucosal fibroids or normal myometrial contraction in the non-pregnant patient. Some helpful findings that characterize adenomyosis include absence of mass effect on the endometrium, poorly defined margins, and on occasion punctate bright signals within the dark mass likely representing islands of deep functioning endometrial tissue and/or foci of hemorrhage.

Dermoid vs. Endometrioma

In addition to the ability to distinguish tissues based on their T1, T2, and hydrogen density differences, MR imaging also allows the distinction of tissues with different chemical composition. Because the resonance frequency of protons of H₂O (water) is different from that of -CH₂ - (fat), it causes either chemical shift artifacts or allows the suppression of signal from one or the other molecule, or allows the acquisition of in- or opposed-phase gradient echo images. This capability allows the easy recognition of fat containing tissues that may appear bright on T1-weighted images.

Dermoid tumors, predominantly fatty, usually contain water-filled components producing internal chemical shift artifacts. Endometriomas on the other hand are blood-

containing cysts and do not contain fat. Although both of these entities may have similar signals on MR imaging, it is the presence or absence of internal chemical shift artifacts and/or the behavior of the mass on fat suppression or opposed-phase gradient echo sequences that clinches the diagnosis. Note however that in a small percentage of mature teratomas, fat may be only detectable on histology and not recognized on MR imaging.

Endometrioma vs. Hemorrhagic Cyst

The differentiation between endometriomas and hemorrhagic cysts is more difficult but clinically not as important. One distinguishing feature is that endometriomas typically demonstrate a gravitational signal gradient due to layering of the complex fluid content. Also, endometriomas are typically multiple. Individual lesions may vary in size, ranging from small (grape-like) to large, and may vary in signal on both T1 and T2-weighted sequences owing to different ages of internal hemorrhage. In contradistinction, hemorrhagic cysts tend to be solitary, homogeneous, and have uniformly hyperintense signal on T2. If differentiation between these two benign entities is clinically important, recommend a follow up US, as endometriomas tend to be stable or grow while hemorrhagic cysts should resolve.

Hemorrhagic Lesion vs. Ovarian Neoplasm

Regardless of whether a hemorrhagic lesion is an endometrioma or a hemorrhagic cyst, the presence of blood products within an ovarian mass essentially excludes an ovarian neoplasm. In theory a mucinous ovarian neoplasm may have bright signal on T1 but in our experience this is extremely rare and an ovarian lesion that is bright on T1 is almost invariably benign.

Obstetrics

MR is ideally suited for the evaluation of maternal disease during pregnancy. The assessment of the uterus and placenta can be easily accomplished in cases of abruption, previa, or suspected placenta accreta. The assessment of the fetus is limited to those cases where sonography is unable to adequately visualize fetal anatomy or when lesions are complex. Since the basic technical limitation of MR is fetal motion, faster sequences are required for adequate visualization of fetal anatomy. Fetal tissues assume similar signals as the adult counterpart except for the fluid-filled fetal lungs that appear bright on T2 weighted images. Both T1- and T2-weighted images are helpful. The single shot or HASTE sequences are ideally suited for assessing fetal tissues, including the brain. MR is also able to distinguish oligohydramnios due to IUGR (no or little subcutaneous fetal fat) from renal agenesis (normal fetal fat).

Malignancies

Uterus/Cervix

Because of the dark signal of the junctional zone on T2-weighted images, and because endometrial and cervical carcinoma have intermediate to high signal intensity

on T2 weighted images, MR depicts degree of invasion into the body of the uterus or cervix. Invasion by these lesions has been correlated with increased signal within or loss of the dark junctional zone separating the endocervix or endometrium from the body of the cervix or myometrium. The staging accuracy of endometrial and endocervical carcinoma is between 80-90%. Because of the rich vascular network about the cervix, MR is better able to distinguish parametrial extension from vessels than CT. Further, because of the dark signal of the muscularis of the bladder wall, anterior extension into the bladder wall can be recognized as a disruption of the dark line. Extensions along the supporting ligaments are depicted as thickening on T1. If there is sufficient bulk, the signal may be increased on T2.

Dynamic imaging with gadolinium is often critical for evaluation. Depending on whether the endometrial or cervical mass is midline or off-line, dynamic images should be obtained as true axials (off-midline mass) or true sagittals (midline mass) with respect to the uterus or cervix. Many of the uterine and cervical cancers enhance rapidly during the arterial phase and then may or may not washout on more delayed sequences. The rapid arterial enhancement seems to be related to the aggressiveness of the lesion and may have prognostic information. Delayed images with fat suppression are often helpful to depict infiltration of parametrial fat.

Thickening of the endometrium (thick bright central zone) can represent a uterine cancer, an endometrial polyp, possibly endometrial hyperplasia, or retained blood from cervical stenosis. Gd can exclude blood if the center of the uterus enhances. For this reason, delayed images through the endometrium (ideally in the sagittal and true axial planes) should always be obtained when gadolinium is given in female pelvis cases. At this time it is not clear whether the enhancement pattern with Gd can distinguish among the other three entities. It has been our experience that endometrium is indistinguishable from endometrial polyps while cancer fails to enhance to the same degree as endometrium on delayed images (**RFM: ?**).

Leiomyosarcoma of the uterus probably begins as a malignant lesion rather than develop within an already existing leiomyoma. Such lesions are heterogeneous on both T1 and T2 weighted sequences. Any bright lesion involving the uterine wall in a postmenopausal woman should be considered malignant.

Ovary

Ovarian carcinoma is easily imaged and recognized as an adnexal mass. The tissue characterization potential of MR, being better than that of US, allows the distinction of fibroids from adnexal masses and the recognition with high confidence of dermoid and endometrioma lesions from ovarian cancers. Gd enhancement has aided in distinguishing malignant from benign lesions when these lesions assume water-signal characteristics. Malignant lesions have enhancing nodular septae, walls, or solid components. For staging and follow up of ovarian neoplasm, MR performed with fat-suppression and with breath-hold sequences appears to be the most sensitive tool for the detection of peritoneal implants. This is likely due to the high sensitivity of MRI to small quantities of contrast.

Male Pelvis

Prostate

Utilizing the body coil, MR imaging can show intrinsic disease, particularly at high field owing to greater T2-weighting. The gland is homogeneous and of uniform signal on T1-weighted images which depict the capsular margin with fat and demonstrate the integrity of the neurovascular bundle. It is however the T2weighted images that allow the distinction of intrinsic zonal anatomy as well as allow the distinction of periprostatic veins from transcapsular extension. Although body coil imaging can display the prostate adequately, an endorectal coil is required for proper staging. Phased-array coils are superior to body-coil imaging to stage prostate cancer but are inferior in performance to the endorectal coil. The most optimal configuration is to use the endorectal coil as part of the phased-array system.

Since we are rarely asked to image the prostate for non-cancerous conditions, all prostate imaging should be done with the endorectal probe. PSA levels generally correlate with cancer burden and extension, but they should not be used as the sole predictor of stage of disease and prognosis as cancer with extensive extension may have low PSA and high PSA (>25) may be present without extension.

Imaging Technique

Patient preparation

Patient should be NPO for at least 3 hours and should be at least 10 to 14 days post biopsy. This time delay is helpful to decrease patient discomfort but is insufficient to eliminate the effect of intraprostatic blood. Blood has been present as late as 5 to 6 weeks post biopsy but rarely after 3 months. Because we are rarely asked to image before biopsy, we have to learn to deal with blood.

The study is best done immediately post-voiding. With the patient in the decubitus position, the coil is inserted such that the coil is facing anteriorly. Inflate the balloon with 80ml and tug back on the balloon against the sphincter to improve the visualization of the prostatic apex.

Pulsing Sequences

Coronal and sagittal images of the retroperitoneum, pelvis, and spine are done to stage lymph nodes and bones for metastases and the kidneys for hydronephrosis. Small field of view T1 and T2w TSE non-breathhold images (with 2 averages) are then done as true coronals and true axials through the prostate. At times with difficult prostate/seminal vesicle anatomy, a sagittal T1 and T2 are added. The study takes approximately 45-60 minutes. See protocol handout for details.

Anatomy

The peripheral zone of the gland is bright because of its glandular structure and high water content. Numerous thin dark strands can be seen coursing from the capsule towards the central region. The periurethral zone is dark owing to its fibromuscular composition. The transitional zone is separated from the peripheral zone by the surgical capsule, is intermediate in signal, and often contains hyperplastic nodules. The surgical capsule is seen in most men, is dark in signal, and allows easy separation of the peripheral zone from the central region. The anterior fibromuscular zone is lens-shaped and is dark. Its lateral margins extend to become part of the outer prostate margin. The latter is made up of the true prostatic capsule (only a few cells deep) and a thick smooth muscle layer. The prostate margin "capsule" is dark on MR owing to the smooth muscle layer. Note that chemical shift artifact may increase the thickness and give a false sense of security on one side of the gland.

The seminal vesicles are paired and abut the superior surface of the peripheral zone of the prostate. Each gland is a convoluted tube typically filled with fluid. In less sexually active men and in the elderly, little fluid may be present. The appearance of the normal seminal vesicle is intermediate on T1 and bright on T2. The thin dark wall of the tube can be seen on T2. The vas deferens is superior, anterior and medial to the seminal vesicles as they join the prostate. It joins the seminal vesicle duct to form the ejaculatory duct, which is recognized on MR when it is surrounded by the peripheral zone. The vas deferens have thick accordion-like dark walls on T2. The ejaculatory duct complex is dark on T2, is a paired structure, and is closely apposed to the surgical capsule as it travels to the verumontanum. At the base, the ejaculatory duct complex may be surrounded by peripheral zone. At times, the tiny bright lumen can be seen within each of the 2 ejaculatory ducts.

Diagnosis

Benign nodules typically seen in the transition zone have mixed heterogeneous signal but may mimic the signal of cancerous lesions. While cancer and benign prostatic hypertrophy may have similar signals in the central region, when cancerous lesions occur in the peripheral zone they appear dark relative to the normally bright peripheral zone.

Hemorrhage in the peripheral zone has severely impacted our ability to test the true detection capability of the technique as blood assumes nearly any combination of signals when viewed on T1- and T2-weighted images potentially camouflaging as well as mimicking cancer. The normal peripheral zone is bright because of the high fluid content within the lumen of the glands. Any pathophysiologic mechanism that decreases the water content in the gland will decrease signal and mimic cancer. Such conditions include glandular atrophy, glandular or stromal hyperplasia which decreases the glandular lumen volume, and granulomatous lesions. Scarring from prior infection or infarction have produced distinct dark linear abnormalities that can be distinguished from focal cancer. Some investigators have used dynamic Gd-enhanced sequences to

assess the time intensity curve or hydrogen-spectroscopy to distinguish between cancer and other conditions that mimic cancer. Early data are encouraging.

Staging

Despite the maturity of CT, it has been shown to be reasonably insensitive in staging prostate cancer with a reported accuracy less than 60%. While transrectal sonography can depict intrinsic prostatic abnormalities, its sensitivity and specificity remain disappointingly low. Certainly, its ability to stage prostate cancer is limited to the capsule. MR imaging, using the body coil, was shown to be accurate in staging cancer with sensitivity and specificity near 70%. To fully stage cancer, two orthogonal T2-weighted sequences are needed. True coronal and axial orientations to the long axis of the prostate are preferred since they allow right and left comparison. Utilizing specialized endorectal coils, others and we have shown a remarkable ability to stage disease that is superior to that achieved with the body coil ranging from 80-90%. Over 90% of extensions will occur at the neurovascular bundles. These are at the 4 and 8 o'clock positions. While both appear dark on T1 the veins are bright and the extension is dark relative to the fat on T2. It should also be noted that the neurovascular bundle moves laterally and anteriorly as it travels towards the apex allowing extension to proceed laterally. Apical cancers have a greater propensity for extension. Therefore, when lesions involve the apex, greater scrutiny of the gland and adjacent muscles is needed.

On high-resolution images, the lumen of the seminal vesicle, even when empty, can still be seen. Regardless of their signal on T2-weighting, asymmetry in signal is abnormal. In such cases, it is the dark gland that is suspected of invasion. Accumulation of blood in the seminal vesicle following needle biopsy is frequent. The presence of blood in the seminal vesicle typically brightens its signal on T1 and may brighten or darken signal on T2-weighted images. Even when blood is dark on T2, the bright signal on T1 can help evaluate the seminal vesicle. The presence of a lesion at the base that is continuous with the seminal vesicle abnormality is the best index for extension. Given that cancer spreads by contiguous extension the presence of intervening normal tissue decreases the suspicion of extension. IV contrast has aided in the assessment of the seminal vesicles.

Scrotum

Currently, high-resolution sonography is the imaging modality of choice because of its excellent depiction of scrotal anatomy, low cost, short examination time and its lack of ionizing radiation. Color Doppler has added anatomic and functional details to evaluate the testis and its surrounding structures. MR imaging is a powerful tool in the assessment of scrotal disease, primarily because it matches or exceeds the sensitivity of sonography but boasts a very high specificity. Like sonography, it is multiplanar, displays flow, and is non-ionizing. However, MR imaging offers superior contrast and spatial resolution and a wide field of view. Its disadvantages include higher cost, limited patient access, and the necessity to sedate boys less than 8 years of age. Presently,

MR is reserved for patients in whom a discrepancy between the clinical assessment and the sonographic findings exists or when sonography is equivocal or non-specific.

Imaging Technique

The patient is placed supine on the scan table feet first. The scrotum is elevated such that the testes lie in a horizontal plane. A 12.5-cm circular multipurpose surface coil or a flex coil is centered over the scrotum and placed horizontally on a 1 cm standoff. The coil is positioned such that the bottom of the coil is over the caudal tip of the scrotal sac. The entire area is then wrapped with the table to minimize patient motion. Ensure that the penis is angled to the side.

Axial and coronal T1 and T2 weighted sequences are acquired with a 16cm FOV and 3-mm slice thickness. Series are obtained from the inferior tip of the scrotum to the inguinal canals and the posterior wall of the scrotum to the anterior abdominal wall. Flow and fat saturation pulses are not needed.

Studies have not yet established whether the use of IV contrast is beneficial in scrotal imaging. With IV contrast, the normal testis and epididymis enhance homogeneously and to a moderate degree. Enhancement persists for a long duration (>10min). Since the agent is eliminated more rapidly from tumors, they become more apparent post contrast. However, there is loss of specificity in that seminomatous and non-seminomatous lesions assume a similar appearance. The use of IV contrast does aid in distinguishing cystic from solid lesions and allows the assessment of testicular vascularity. Although the enhancement is usually homogeneous, inhomogeneous enhancement patterns have been observed mostly likely secondary to ischemic injury or fibrosis.

Anatomy

The normal testis is a sharply demarcated oval structure with low to intermediate signal intensity on the T1-weighted images and high homogeneous signal intensity on the T2-weighted images. It is surrounded by the tunica albuginea that has low signal intensity on T2-weighted images. The small amount of fluid present between the layers of the tunica vaginalis follows the expected signal behavior of water. The mediastinum testis assumes similar signal as testis on the T1-weighted sequence and becomes darker than testis on the T2-weighted sequence. Intrinsic testicular signal, although homogeneous, displays the rete testes that radiate from the mediastinum to the tunical surface.

The epididymis is intermediate in signal to fat and testis on T2. It is somewhat heterogeneous in signal. The head of the epididymis and at times the tail can be surrounded by fluid. The scrotal wall is dark on T2. Superior and lateral to the upper pole of the testis is the pampiniform plexus that is heterogeneous in signal on both T1 and T2.

Tumors

The overall MR appearance of testicular tumors is dependent upon their histology. Seminomatous lesions are isointense with testis on T1- and darker on T2-weighted images and are somewhat homogeneous, and clearly demarcated from normal testicular tissue. Non-seminomatous lesions are heterogeneous in signal on both T1- and T2-weighted images owing to their complex histology. The overall intensity of most non-seminomatous lesions is bright, almost equal to that of testis. A dark fibrous tumor capsule typically surrounds these lesions. The ability of MRI to distinguish seminomatous from non-seminomatous lesions is 93%.

Benign Lesions

The appearance of dilated intratesticular seminiferous tubules has been recognized and described on both sonography and MR. The findings are somewhat characteristic on sonography, potentially allowing a specific diagnosis to be made, thereby obviating the need for surgery. This intratesticular process is associated with large ipsilateral spermatoceles, is centered at the mediastinum testis, and is contiguous with the body of the epididymis. The mass of ectatic tubules at the mediastinum is homogeneous and of lower signal on T1- and isointense or slightly brighter than testis on T2-weighted images and is identical to that of the ipsilateral spermatoceles on all sequences. Because the tubules do not enhance with Gd, their conspicuity and tubular pattern become apparent following IV contrast.

For further reading

Liver & Biliary tree

- Reimer P, Jahnke N, Fiebich M, Schima W, Deckers F, Marx C, Holzknrecht N, Saini S. Hepatic lesion detection and characterization: value of nonenhanced MR imaging, superparamagnetic iron oxide-enhanced MR imaging, and spiral CT-ROC analysis. *Radiology* 2000 Oct;217(1):152-8
- Ichikawa T, Federle MP, Grazioli L, Marsh W. Fibrolamellar hepatocellular carcinoma: pre- and posttherapy evaluation with CT and MR imaging. *Radiology* 2000 Oct;217(1):145-51
- Ito K, Siegelman ES, Stolpen AH, Mitchell DG. MR imaging of complications after liver transplantation. *AJR* 2000 Oct;175(4):1145-9
- Mortelet KJ, Praet M, Van Vlierberghe H, Kunnen M, Ros PR. CT and MR imaging findings in focal nodular hyperplasia of the liver: radiologic-pathologic correlation. *AJR* 2000 Sep;175(3):687-92
- Vitellas KM, Keogan MT, Freed KS, Enns RA, Spritzer CE, Baillie JM, Nelson RC. Radiologic manifestations of sclerosing cholangitis with emphasis on MR cholangiopancreatography. *Radiographics* 2000 Jul-Aug;20(4):959-75
- Ward J, Guthrie JA, Scott DJ, Atchley J, Wilson D, Davies MH, Wyatt JI, Robinson PJ. Hepatocellular carcinoma in the cirrhotic liver: double-contrast MR imaging for diagnosis. *Radiology* 2000 Jul;216(1):154-62
- Ichikawa T, Federle MP, Grazioli L, Madariaga J, Nalesnik M, Marsh W. Fibrolamellar hepatocellular carcinoma: imaging and pathologic findings in 31 recent cases. *Radiology* 1999;213(2):352-61
- Semelka RC, Cance WG, Marcos HB, Mauro MA. Liver metastases: comparison of current MR techniques and spiral CT during arterial portography for detection in 20 surgically staged cases. *Radiology* 1999 Oct;213(1):86-91
- Schultz JF, Bell JD, Goldstein RM, Kuhn JA, McCarty TM. Hepatic tumor imaging using iron oxide MRI: comparison with computed tomography, clinical impact, and cost analysis. *Ann Surg Oncol* 1999;6(7):691-8
- Worawattanakul S, Semelka RC, Noone TC, Calvo BF, Kelekis NL, Woosley JT. Cholangiocarcinoma: spectrum of appearances on MR images using current techniques. *Magn Reson Imaging* 1998;16(9):993-1003
- Fulcher AS, Turner MA. Pitfalls of MR cholangiopancreatography (MRCP). *J Comput Assist Tomogr* 1998;22(6):845-50
- Ferrucci JT. Advances in abdominal MR imaging. *Radiographics* 1998; 18(6):1569-86
- Yu JS, Kim MJ, Kim KW, Chang JC, Jo BJ, Kim TH, Lee JT, Yoo HS. Hepatic cavernous hemangioma: monographic patterns and speed of contrast enhancement on multiphase dynamic MR imaging. *AJR* 1998; 171(4):1021-5
- Kawamoto S, Soyer PA, Fishman EK, Bluemke DA. Nonneoplastic liver disease: evaluation with CT and MR imaging. *Radiographics* 1998;18(4):827-48
- Murakami T, Kim T, Nakamura H. Hepatitis, cirrhosis, and hepatoma. *JMRI* 1998;8(2):346-58
- Stafford-Johnson DB, Hamilton BH, Dong Q, Cho KJ, Turcotte JG, Fontana RJ, Prince MR. Vascular complications of liver transplantation: evaluation with gadolinium-enhanced MR angiography. *Radiology* 1998;207(t):153-60
- Outwater EK, Ito K, Siegelman E, Martin CE, Bhatia M, Mitchell DG. Rapidly enhancing hepatic hemangiomas at MRI: distinction from malignancies with T2-weighted images. *JMRI* 1997;7(6):1033-9
- Leyendecker JR, Rivera E Jr, Washburn WK, Johnson SP, Diffin DC, Eason JD. MR angiography of the portal venous system: techniques, interpretation, and clinical applications *Radiographics* 1997;17(6):1425-43

- Fulcher AS, Turner MA. HASTE MR cholangiography in the evaluation of hilar cholangiocarcinoma. *AJR* 1997 Dec; 169(6):1501-5
- Gabata T, Matsui O, Kadoya M, Yoshikawa J, Ueda K, Kawamori Y, Takashima T. Segmental hyperintensity on T1-weighted MRI of the liver: indication of segmental cholestasis. *JMRI* 1997;7(5):855-7
- Mathieu D, Paret M, Mahfouz AE, Caseiro-Alves F, Tran Van Nhieu J, Anglade MC, Rahmouni A, Vasile N. Hyperintense benign liver lesions on spin-echo T1-weighted MR images: pathologic correlations. *Abdom Imaging* 1997;22(4):410-7
- Paley MR, Ros PR. MR imaging of the liver--a practical approach. *Magn Reson Imaging Clin N Am* 1997;5(2):415-29
- Lee MG, Lee HJ, Kim MH, Kang EM, Kim YH, Lee SG, Kim PN, Ha HK, Auh YH. Extrahepatic biliary diseases: 3D MR cholangiopancreatography compared with endoscopic retrograde cholangiopancreatography. *Radiology* 1997;202(3):663-9
- MS, Baron RL, Murakami T. Hepatic malignancies: usefulness of acquisition of multiple arterial and portal venous phase images at dynamic gadolinium-enhanced MR imaging. *Radiology* 1996 Nov; 201(2):337-45.
- Rubin RA, Mitchell DG. Evaluation of the solid hepatic mass. [Review] *Medical Clinics of North America* 1996 Sep; 80(5):907-28.
- Sencenterre E, Taourel P, Bouvier Y, Pradel J, Van Beers B, Daures JP, Pringot J, Mathieu D, Bruel JM. Detection of hepatic, metastases: ferumoxides-enhanced MR imaging versus unenhanced MR imaging and CT during arterial portography. *Radiology* 1996 Sep; 200(3):785-92.
- Gaa J, Hatabu H, Jenkins PL, Finn JP, Edelman RR. Liver masses: replacement of conventional T2-weighted spin-echo MR imaging with breath-hold MR imaging. *Radiology* 1996 Aug; 200(2):459-64.
- Zerhouni EA, Rutter C, Hamilton SR, Balfe DM, Megibow AJ, Francis IR, Moss AA, Heiken JP, Tempany CM, Aisen AM, Weiareb JC, Gatsonis C, McNeil BJ. CT and MR imaging in the staging of colorectal carcinoma: report of the Radiology Diagnostic Oncology Group 11. *Radiology* 1996 Aug; 200(2):443-5 1.
- Caudana R, Morana G, Pirovano GP, Nicoli N, Portuese A, Spinazzi A, Di Rito R, Pistolesi GF. Focal malignant hepatic lesions: MR imaging enhanced with gadolinium benzyloxypropionictetra-acetate (BOPTA)--preliminary results of phase 11 clinical application. *Radiology* 1996 May; 199(2):513-20.
- Murakami T, Baron RL, Peterson MS, Oliver JH 3rd, Davis PL, Confer SR, Federle MP. Hepatocellular carcinoma: MR imaging with mangafodipir trisodium (Mn-DPDP). *Radiology* 1996 Jul; 200(i):69-77.
- Miyazaki T, Yamashita Y, Tsuchigame T, Yamamoto H, Urata J, Takahashi M. MR cholangiopancreatography using HASTE (half-Fourier acquisition single-shot turbo spin echo) sequences. *AJR* 1996 Jun; 166(6):1297-1303.
- Chung KY, Mayo Smith WW, Saini S, Rahmouni A, Goili M, Mathieu D. Hepatocellular adenoma: MR imaging features with pathologic correlation. *AJR* 1995 Aug; 165 (2):303-8.
- Paulson EK, McClellan JS, Washington K, Spritzer CE, Meyers WC, Baker ME. Hepatic adenoma: MR characteristics and correlation with pathologic findings. *AJR* 1994 Jul; 163 (1):113-6.
- Whitney WS, Herfkens RJ, Jeffrey R-B, McDonnell CH, Li KC, Van Dalsem WJ, Low RN, Francis [R, Dabatin JF, Glazer GM. Dynamic breath-hold multiplanar spoiled gradient recalled MR imaging with gadolinium enhancement for differentiating hepatic hemangiomas from malignancies at 1.5 T. *Radiology* 1993 Dec; 189(3):863-70.

Pancreas

- Adamek HE, Albert J, Breer H, Weitz M, Schilling D, Riemann JF. Pancreatic cancer detection with magnetic resonance cholangiopancreatography and endoscopic retrograde cholangiopancreatography: a prospective controlled study. *Lancet* 2000 Jul 15;356(9225):190-3

- Meador TL, Krebs TL, Cheong JJ, Daly B, Keay S, Bartlett S. Imaging features of post transplantation lymphoproliferative disorder in pancreas transplant recipients. *AJR* 2000; 174(i):121-4
- Romijn MG, Stoker J, van Eijck CH, van Muiswinkel JM, Torres CG, Lameris JS. MRI with mangafodipir trisodium in the detection and staging of pancreatic cancer. *J Magn Reson Imaging* 2000 Aug;12(2):261-8
- Ito K, Mitchell DG, Outwater EK. Pancreatic changes in primary sclerosing cholangitis: evaluation with MR imaging. *AJR* 1999; 173(6):1535-9
- Matos C, Nicaise N, Metens T, Cremer M, Deviere J. Secretin-enhanced MR pancreatography. *Semin Ultrasound CT MR* 1999;20(5):340-51
- Catalano C, Pavone P, Laghi A, Panebianco V, Scipioni A, Fanelli F, Brillo R, Passariello R. Pancreatic adenocarcinoma: combination of MR imaging, MR angiography and MR cholangiopancreatography for the diagnosis and assessment of resectability. *Eur Radiol* 1998;8(3):428-34
- Buctow PC, Rao P, Thompson LD. From the Archives of the AFIP. Mucinous cystic neoplasms of the pancreas: radiologic-pathologic correlation. *Radiographics* 1998; 18(2):433-49
- Sho M, Nakajima Y, Kanchiro H, Hisanaga M, Nishio K, Nagao M, Tatekawa Y, Ikeda N, Kanokogi H, Yamada T, Hirohashi S, Hirohashi R, Uchida H, Nakano H. A new evaluation of pancreatic function after pancreatoduodenectomy using secretin magnetic resonance cholangiopancreatography. *Am J Surg* 1998; 176(3):279-82
- Hidaka T, Hirohashi S, Uchida H, Koh M, Itoh T, Matsuo Y, Matsuo N, Ohishi H. Annular pancreas diagnosed by single-shot MR cholangiopancreatography. *Magn Reson Imaging* 1998; 16(4):441-4
- Matos C, Deviere J, Cremer M, Nicaise N, Struyven J, Metens T. Acinar filling during secretin-stimulated MR pancreatography. *AJR* 1998; 171(t):165-9
- Mayo-Smith WW, Schima W, Saini S, Slater GJ, McFarland EG. Pancreatic enhancement and pulse sequence analysis using low-dose mangafodipir trisodium. *AJR* 1998; 170(3):649-52
- Sugiyama M, Atomi Y, Hachiya J. Intraductal papillary tumors of the pancreas: evaluation with magnetic resonance cholangiopancreatography. *Am J Gastroenterol* 1998;93(2):156-9
- frie H, Honda H, Kaneko K, Kuroiwa T, Yoshimitsu K, Masuda K. Comparison of helical CT and MR imaging in detecting and staging small pancreatic adenocarcinoma. *Abdom Imaging* 1997;22(4):429-33
- Matos C, Metens T, Deviere J, Nicaise N, Braude P, Van Yperen G, Cremer M, Struyven J. Pancreatic duct: morphologic and functional evaluation with dynamic MR pancreatography after secretin stimulation. *Radiology* 1997;203(2):435-41
- Buetow PC, Buck JL, Pantongrag Brown L, Beck KG, Ros PR, Adair CF. Solid and papillary epithelial neoplasm of the pancreas: imaging-pathologic correlation on 56 cases. *Radiology* 1996 Jun; 199(3):707-11
- Megibow AJ, Zhou XH, Rotterdam H, Francis IR, Zerhouni EA, Balfe DM, Weinreb JC, Aisen A, Kuhlman J, Heiken JP, et al. Pancreatic adenocarcinoma: CT versus MR imaging in the evaluation of resectability--report of the Radiology Diagnostic Oncology Group. *Radiology* 1995 May; 195(2):327-32.
- Gabata T, Matsui O, Kadoya M, Yoshikawa J, Miyayama S, Takashima T, Nagakawa T, Kayahara M, Nonomura A. Small pancreatic adenocarcinomas: efficacy of MR imaging with fat suppression and gadolinium enhancement. *Radiology* 1994 Dec; 193(3):683-8.

Kidney

- Halpern EJ, Mitchell DG, Wechsler RJ, Outwater EK, Moritz MJ, Wilson GA. Preoperative evaluation of living renal donors: comparison of CT angiography and MR angiography. *Radiology* 2000 Aug;216(2):434-9

- Krestin GP. Genitourinary MR: kidneys and adrenal glands. *Eur Radiol* 1999;9(9):1705-14 Dong Q, Schoenberg SO, Carlos RC, Neimatallah M, Cho KJ, Williams DM, Kazaniian SN, Prince MR. Diagnosis of renal vascular disease with MR angiography. *Radiographics* 1999; 19(6):1535-54
- Prince MR. Renal MR angiography: a comprehensive approach. *JMRI* 1998;8(3):511-6
- Zagoria RJ, Dyer PB. The small renal mass: detection, characterization, and management. *Abdom Imaging* 1998;23(3):256-65
- Newhouse JH, Wagner BJ. Renal oncocytomas. *Abdom Imaging* 1998;23(3):249-55
- Bakker J, Beek FJ, Beutler JJ, Hene RJ, de Kort GA, de Lange EE, Moons KG, Mali WP. Renal artery stenosis and accessory renal arteries: accuracy of detection and visualization with gadolinium-enhanced breath-hold MR angiography. *Radiology* 1998;207(2):497-504
- Niendorf ER, Grist TM, Lee FT Jr, Brazy PC, Santyr GE. Rapid in vivo measurement of single-kidney extraction fraction and glomerular filtration rate with MR imaging. *Radiology* 1998; 206(3):791-8
- Choyke PL, Walther MM, Wagner JR, Rayford W, Lyne JC, Linehan WM. Renal cancer: preoperative evaluation with dual-phase three-dimensional MR angiography. *Radiology* 1997;205(3):767-71
- Jinzaki M, Tanimoto A, Narimatsu Y, Ohkuma K, Kurata T, Shinmoto H, Hiramatsu K, Mukai M, Murai M. Angiomyolipoma: imaging findings in lesions with minimal fat. *Radiology* 1997;205(2):497-502
- Prince MR, Schoenberg SO, Ward JS, Londy FJ, Wakefield TW, Stanley JC. Hemodynamically significant atherosclerotic renal artery stenosis: MR angiographic features. *Radiology* 1997 Oct;205(1):128-36
- Outwater EK, Bhatia M, Siegelman ES, Burke MA, Mitchell DG. Lipid in renal clear cell carcinoma: detection on opposed-phase gradient-echo MR images. *Radiology* 1997;205(1):103-7
- Yamashita Y, Honda S, Nishiharu T, Urata J, Takahashi M. Detection of pseudocapsule of renal cell carcinoma with MR imaging and CT. *AJR* 1996 May; 166(5):1151-5
- Semelka RC, Kelekis NL, Burdeny DA, Mitchell DG, Brown JJ, Siegelman ES. Renal lymphoma: demonstration by MR imaging. *AJR* 1996 Apr; 166(4):823-7.

Adrenal Glands

- Barzon L, Boscaro M. Diagnosis and management of adrenal incidentalomas. *J Urol* 2000; 163(2):398-407
- Krebs TL, Wagner BJ. MR imaging of the adrenal gland: radiologic-pathologic correlation. *Radiographics* 1998;18(6):1425-40
- Choyke PL. From needles to numbers: can noninvasive imaging distinguish benign and malignant adrenal lesions? *World J Urol* 1998; 16(1):29-34
- Jalil ND, Pattou FN, Combemale F, Chapuis Y, Henry JF, Peix JL, Proye CA. Effectiveness and limits of preoperative imaging studies for the localization of pheochromocytomas and paragangliomas: a review of 282 cases. French Association of Surgery (AFC), and The French Association of Endocrine Surgeons (AFCE). *Eur J Surg* 1998; 164:23-8
- Gilfeather M, Woodward PJ. MR imaging of the adrenal glands and kidneys, *Semin Ultrasound CT MR* 1998; 19(1):53-66
- Schwartz LH, Ginsberg MS, Burt ME, Brown KT, Getrajdman GI, Panicek DM. MRI as an alternative to CT-guided biopsy of adrenal masses in patients with lung cancer. *Ann Thorac Surg* 1998;65(1):193-7
- Korobkin M, Francis IR. Imaging of adrenal masses. *Urol Clin North Am* 1997;24(3):603-22
- Korobkin M, Lombardi TJ, Aisen AM, Francis IR, Quint LE, Dunnick NR, Londy F, Shapiro B, Gross MD, Thompson NW. Characterization of adrenal masses with chemical shift and gadolinium-enhanced MR imaging. *Radiology* 1995 Nov; 197(2):411-8.
- Outwater EK, Siegelman ES, Radecki PD, Piccoli CW, Mitchell DG. Distinction between benign and malignant adrenal masses: value of T1-weighted chemical-shift MR imaging. *AJR* 1995 Sep; 165(3):579-83.
- Mayo Smith WW, Lee MJ, McNicholas MM, Hahn PF, Boland GW, Saini S. Characterization of adrenal masses (< 5 cm) by use of chemical shift MR imaging: observer performance versus quantitative measures. *AJR* 1995 Jul; 165:91-5.

- Tsushima Y, Ishizaka H, Matsumoto M. Adrenal masses: Differentiation with chemical shift, FLASH imaging. *Radiology* 1993; 186:705-709.

Lymphnodes

- Boieselle PM, Patz EF Jr, Vining DJ, Weissleder R, Shepard JA, McCloud TC. Imaging of mediastinal lymph nodes: CT, MR, and FDG PET. *Radiographics* 1998; 18(5):1061-9
- Bellin MF, Roy C, Kinkel K, et al. Lymph node metastases: safety and effectiveness of MR imaging with ultrasmall superparamagnetic iron oxide particles--initial clinical experience. *Radiology* 1998;207(3):799-808
- Scheidler J, Hricak H, Yu KK, Subak L, Segal MR. Radiological evaluation of lymph node metastases in patients with cervical cancer. A meta-analysis. *JAMA* 1997;278:1096- 1 01
- Anzai Y, Brunberg JA, Lufkin RB. Imaging of nodal metastases in the head and neck. *JMRI* 1997;7(5):774-83
- Mussurakis S, Buckley DL, Horsman A. Prediction of axillary lymph node status in invasive breast cancer with dynamic contrast-enhanced MR imaging. *Radiology* 1997;203:3 17-21
- Weinreb JC, Naidich DP. Thoracic magnetic resonance imaging. *Clin Chest Med* 1991; 12:33-54.
- Jabour BA, Lufkin RB, Layfield LJ, Hanafee VN. Magnetic resonance imaging of metastatic cervical adenopathy. *JMRI* 1990; 2:69-75.
- Hanun B, Taupitz M, Hussmann P, Wagner S, Wolf KJ. MR lymphography with iron oxide particles: dose-response studies and pulse sequence optimization in rabbits. *AJR* 1992; 183-190.

Female Pelvis

- Postema S, Pattynama PM, van den Berg-Huysmans A, Peters LW, Kenter G, Trimbos JB. Effect of MRI on therapeutic decisions in invasive cervical carcinoma. *Gynecol Oncol* 2000; 79:485-9
- Yamashita Y, Baba T, Baba Y, Nishimura R, Ikeda S, Takahashi M, Ohtake H, Okamura H. Dynamic contrast-enhanced MR imaging of uterine cervical cancer: pharmacokinetic analysis with histopathologic and its importance in predicting the outcome of radiation therapy. *Radiology* 2000;216:803-9
- Li W, Brophy DP, Chen Q, Edelman RR, Prasad PV. Semiquantitative assessment of uterine perfusion using first pass dynamic contrast-enhanced MR imaging for patients treated with uterine fibroid embolization. *J Magn Reson Imaging* 2000; 12:1004-8
- Byun JY, Kim SE, Choi BG, Ko GY, Jung SE, Choi KH. Diffuse and focal adenomyosis: MR imaging findings. *Radiographics* 1999 Oct;] 9 Spec No:S 161-70
- Mayr &A, Hawighorst H, Yuh WT, Essig M, Magnotta VA, Knopp MV. MR microcirculation assessment in cervical cancer: correlations with histomorphological tumor markers and clinical outcome. *JMRI* 1999; 10(3):267-76
- Murase E, Siegelman ES, Outwater EK, Perez-Jaffe LA, Tureck RW. Uterine leiomyomas: histopathologic features, MR imaging findings, differential diagnosis, and treatment. *Radiographics* 1999; 19(5):1179-97
- Lai CH, Hong JH, Hsueh S, Ng KK, Chang TC, Tseng CJ, Chou HH, Huang K. Preoperative prognostic variables and the impact of postoperative adjuvant therapy on the outcomes of Stage IB or II cervical carcinoma patients with or without pelvic lymph node metastases: an analysis of 891 cases. *Cancer* 1999;85(7):1537-46
- Brosens JJ, Barker FG, de Souza NM. Myometrial zonal differentiation and uterine junctional zone hyperplasia in the non-pregnant uterus. *Hum Reprod Update* 1998;4(5):496-502
- Hawighorst H, Schoenberg SO, Knapstein PG, Knopp MV, Schaeffer U, Essig M, van Kaick G. Staging of invasive cervical carcinoma and of pelvic lymph nodes by high resolution MRI with a phased-array coil in comparison with pathological findings. *J Comput Assist Tomogr* 1998;22(1):75-81
- Hawnaur JM, Johnson RJ, Carrington BM, Hunter RD. Predictive value of clinical examination, transrectal ultrasound and magnetic resonance imaging prior to radiotherapy in carcinoma of the cervix. *Br J Radiol* 1998;71(848):819-27

- Postema S, Pattynama PM, Broker S, van der Geest RJ, van Rijswijk CS, Baptist Trimbos J. Fast dynamic contrast-enhanced colour-coded MRI in uterine cervix carcinoma: useful for tumour staging? *Clin Radiol* 1998;53(10):729-34
- Hawighorst H, Weikel W, Knapstein PG, Knopp MV, Zuna 1, Schonberg SO, Vaupel P, van Kaick G. Angiogenic activity of cervical carcinoma: assessment by functional magnetic resonance imaging-based parameters and a histomorphological approach in correlation with disease outcome. *Clin Cancer Res* 1998;4(10):2305-12
- Sundfor K, Lyng H, Rofstad EK. Tumour hypoxia and vascular density as predictors of metastasis in squamous cell carcinoma of the uterine cervix. *Br J Cancer* 1998;78(6):822-7
- Ha TP, Li KC. Placenta accreta: MRI antenatal diagnosis and surgical correlation. *JMRI* 1998;8(3):748-50
- Zreik TG, Troiano RN, Ghoussoub RAD, Olive DL, Arici A, McCarthy SM. Myometrial tissue in uterine septa. *J Am Assoc Gynecol Laparosc* 1998;5(2):155-60
- Van Vierzen PB, Massuger LF, Ruys SH, Barentsz JO. Fast dynamic contrast enhanced MR imaging of cervical carcinoma. *Clin Radiol* 1998;53(3):183-92
- Outwater EK, Siegelman ES, Van Deerlin V. Adenomyosis: current concepts and imaging considerations. *AJR* 1998;170(2):437-41
- Siegelman ES, Outwater EK, Banner MP, Ramchandani P, Anderson TL, Schnall MD. High-resolution MR imaging of the vagina. *Radiographics* 1997; 17(5):1183-203
- Troiano RN, Lazzarini KM, Scoutt LM, Lange RC, Flynn SD, McCarthy S. Fibroma and fibrothecoma of the ovary: MR imaging findings. *Radiology* 1997;204(3):795-8
- Yamashita Y, Hatanaka Y, Torashima M, Takahashi M, Miyazaki K, Okamura H. Characterization of sonographically indeterminate ovarian tumors with MR imaging. A logistic regression analysis. *Acta Radiol* 1997;38(4 Pt 1):572-7
- Kimura 1, Togashi K, Kawakami S, Nakano Y, Takakura K, Mori T, Konishi J. Polycystic ovaries: implications of diagnosis with MR imaging. *Radiology* 1996 Nov; 201(2):549-52.
- Hawighorst H, Knapstein PG, Weikel W, Knopp MV, Schaeffer U, Brix G, Essig M, Hoffmann U, Zuna 1, Schonberg S, van Kaick G. Cervical carcinoma: comparison of standard and pharmacokinetic MR imaging. *Radiology* 1996 Nov; 201(2):531-9.
- Yang WT, Walkden SB, Ho S, Cheung TH, Lam SK, Teo J, Metreweli C. Transrectal ultrasound in the evaluation of cervical carcinoma and comparison with spiral computed tomography and magnetic resonance imaging. *British Journal of Radiology* 1996 Jul; 69(823):610-6.
- Kang S, Turner DA, Foster GS, Rapoport MI, Spencer SA, Wang JZ. Adenomyosis: specificity of 5 mm as the maximum normal uterine junctional zone thickness in MR images. *AJR* 1996 May; 166(5):1145-50
- Brosens JJ, de Souza NM, Barker FG. Uterine junctional zone: function and disease. [Review] *Lancet* 1995; 346:558-60.
- Outwater EK, Dunton CJ. Imaging of the ovary and adnexa: clinical issues and applications of MR imaging. [Review] *Radiology* 1995 Jan; 194(1):1-18.
- Schnall MD. Magnetic resonance evaluation of uterine malignancies. [Review] *Seminars in Ultrasound, CT & MR* 1994 Feb; 15(1):27-37.
- Smith RC, McCarthy S. Magnetic resonance staging of neoplasms of the uterus. *Radiologic Clinics of North America* 1994 Jan; 32(1):109-31.
- Hricak H. Magnetic resonance imaging evaluation of the irradiated female pelvis. [Review] *Seminars in Roentgenology* 1994 Jan; 29(1):70-80.
- Yamashita Y, Hatanaka Y, Torashima M, Takahashi M, Miyazaki K, Okamura H. Mature cystic teratomas of the ovary without fat in the cystic cavity: MR features in 12 cases. *AJR* 1994 Sep; 163(3):613-6.
- Wagner BJ, Woodward PJ. Magnetic resonance evaluation of congenital uterine anomalies. [Review] *Seminars in Ultrasound, CT & MRI* 1994 Feb; 15(1):4-17.
- Riccio TJ, Adams HG, Munzing DE, Mattrey RF: Magnetic Resonance Imaging as an Adjunct to Sonography in the Evaluation of the Female Pelvis. *Magnetic Resonance Imaging*, 1990; 8:699-704.

Obstetrics

- Levine D, Barnes P, Korf B, Edelman R. Tuberos sclerosi in the fetus: second-trimester diagnosis of subependymal tubers with ultrafast MR imaging. *AJR* 2000;175:1067-9
- Coakley FV, Lopoo JB, Lu Y, Hricak H, Albanese CT, Harrison MR, Filly RA. Normal and hypoplastic fetal lungs: volumetric assessment with prenatal single-shot rapid acquisition with relaxation enhancement MR imaging. *Radiology* 2000 Jul;216(1):107-11
- Stazzone MM, Hubbard AM, Bilaniuk LT, Harty MP, Meyer JS, Zimmerman RA, Mahboubi S. Ultrafast MR imaging of the normal posterior fossa in fetuses. *AJR* 2000;175:835-9.
- Wagenvoort AM, Bekker MN, Go AT, Vandenbussche FP, Van Buchem MA, Valk J, Van Vugt JM. Ultrafast scan magnetic resonance in prenatal diagnosis. *Fetal Diagn Ther* 2000;15:364-72
- Palacios Jaraquemada JM, Bruno C. Gadolinium-enhanced MR imaging in the differential diagnosis of placenta accreta and placenta percreta. *Radiology* 2000; 216:610-1
- Hubbard AM, Crombleholme TM, Adzick NS, Coleman BG, Howell LJ, Meyer JS, Flake AW. Prenatal MRI evaluation of congenital diaphragmatic hernia. *Am J Perinatol* 1999;16:407-13
- Huppert BJ, Brandt KR, Ramin KD, King BF. Single-shot fast spin-echo MR imaging of the fetus: a pictorial essay. *Radiographics* 1999 Oct; 19 Spec No:S215-27
- Coakley FV, Hricak H, Filly RA, Barkovich AJ, Harrison MR. Complex fetal disorders: effect of MR imaging on management--preliminary clinical experience. *Radiology* 1999;213(3):691-6
- Maldjian C, Adam R, Pelosi M, Pelosi M 3rd, Rudelli RD, Maidjian J. MRI appearance of placenta percreta and placenta accreta. *Magn Reson Imaging* 1999; 17(7):965-71
- Hubbard AM, Adzick NS, Crombleholme TM, Coleman BG, Howell LJ, Haselgrove JC, Mahboubi S. Congenital chest lesions: diagnosis and characterization with prenatal MR imaging. *Radiology* 1999;212(l):43-8
- Amin RS, Nikolaidis P, Kawashima A, Kramer LA, Ernst RD. Normal anatomy of the fetus at MR imaging. *Radiographics* 1999; 19 Spec No: S201-14
- Maldjian C, Milestone B, Schnall M, Smith R. MR appearance of uterine dehiscence in the post-cesarean section patient. *J Comput Assist Tomogr* 1998;22(5):738-41
- Forstner R, Kalbhen CL, Filly RA, Hricak H. Abdominopelvic MR imaging in the nonobstetric evaluation of pregnant patients. *AJR* 1996 May; 166(5):1139-44

Prostate

- Kurhanewicz J, Vigneron DB, Males RG, Swanson MG, Yu KK, Hricak H. The prostate: MR imaging and spectroscopy. Present and future. *Radiol Clin North Am* 2000;38:115-38
- Jager GJ, Severens JL, Thornbury JR, de La Rosette JJ, Ruijs SH, Barentsz JO. Prostate cancer staging: should MR imaging be used?--A decision analytic approach. *Radiology* 2000;215:445-51.
- D'Amico AV, Whittington R, Malkowicz SB, Wu YH, Chen M, Art M, Tomaszewski JE, Wein A. Combination of the preoperative PSA level, biopsy gleason score, percentage of positive biopsies, and MRI T-stage to predict early PSA failure in men with clinically localized prostate cancer. *Urology* 2000;55:572-7.
- D'Amico AV, Whittington R, Malkowicz B, Schnall M, Schultz D, Cote K, Tomaszewski JE, Wein A. Endorectal magnetic resonance imaging as a predictor of biochemical outcome after radical prostatectomy in men with clinically localized prostate cancer. *J Urol* 2000;164:759-63
- Coakley FV, Hricak H. Radiologic anatomy of the prostate gland: a clinical approach. *Radiol Clin North Am* 2000; 38:15-30
- Yu KK, Scheidler J, Hricak H, Vigneron DB, Zaloudek CJ, Males RG, Nelson SJ, Carroll PR, Kurhanewicz J. Prostate cancer: prediction of extracapsular extension with endorectal MR imaging and three-dimensional proton MR spectroscopic imaging. *Radiology* 1999;213(2):481-8
- Scheidler J, Hricak H, Vigneron DB, Yu KK, Sokolov DL, Huang LR, Zaloudek CJ, Nelson SJ, Carroll PR, Kurhanewicz J. Prostate cancer: localization with three-dimensional proton MR spectroscopic imaging--clinicopathologic study. *Radiology* 1999;213(2):473-80
- Barentsz JO, Engelbrecht M, Jager GJ, Witjes JA, de LaRosette J, van Der Sanden BP, Huisman HJ, Heerschap A. Fast dynamic gadolinium-enhanced MR imaging of urinary bladder and prostate cancer. *JMRI* 1999; 10(3):295-304

- Kim JK, Kim DY, Lee YH, Sung NK, Chung DS, Kim OD, Kim KB. *In vivo* differential diagnosis of prostate cancer and benign prostatic hyperplasia: localized proton magnetic resonance spectroscopy using external-body surface coil. *Magn Reson Imaging* 1998; 16(10):1281-8
- Manzone TA, Malkowicz SB, Tomaszewski JE, Schnall MD, Langlotz CP. Use of endorectal MR imaging to predict prostate carcinoma recurrence after radical prostatectomy. *Radiology* 1998;209(2):537-42
- Myers RP, Cahill DR, Devine RM, King BF. Anatomy of radical prostatectomy as defined by magnetic resonance imaging. *J Urol* 1998; 159(6):2148-58
- D'Amico AV, Schnall M, Whittington R, Malkowicz SB, Schultz D, Tomaszewski JE, Wein A. Endorectal coil magnetic resonance imaging identifies locally advanced prostate cancer in select patients with clinically localized disease. *Urology* 1998;51(3):449-54
- Kaji Y, Kurhanewicz J, Hricak H, Sokolov DL, Huang LR, Nelson SJ, Vigneron DB. Localizing prostate cancer in the presence of postbiopsy changes on MR images: role of proton MR spectroscopic imaging. *Radiology* 1998;206:785-90
- Ravery V, Boccon-Gibod L. T3 prostate cancer: how reliable is clinical staging? *Semin Urol Oncol* 1997; 15(4):202-6
- Parsons RB, Fisher AM, Bar-Chama N, Mitty HA. MR imaging in male infertility. *Radiographics* 1997;17(3):627-37
- Yu KK, Hricak H, Alagappan R, Chernoff DM, Bacchetti P, Zaloudek CJ. Detection of extracapsular extension of prostate carcinoma with endorectal and phased-array coil MR imaging multivariate feature analysis. *Radiology* 1997;202(3):697-702
- Tempany CM, Zhou X, Zerhouni EA, Rifkin MD, Quint LE, Piccoli CW, Ellis JH, McNeil BJ. Staging of prostate cancer: results of Radiology Diagnostic Oncology Group project comparison of three MR imaging techniques. *Radiology*. 192(1):47-54, 1994
- Ellis JH, Tempany C, Sarin MS, Gatsonis C, Rifkin MD, McNeil BJ. MR imaging and sonography of early prostatic cancer: pathologic and imaging features that influence identification and diagnosis. *AJR*. 162(4):865-72, 1994

Scrotum

- Watanabe Y, Dohke M, Ohkubo K, Ishimori T, Amoh Y, Okumura A, Oda K, Hayashi T, Dodo Y, Arai Y. Scrotal disorders: evaluation of testicular enhancement patterns at dynamic contrast-enhanced subtraction MR imaging. *Radiology* 2000; 217:219-27
- Rouviere O, Bouvier R, Pangaud C, Jeune C, Dawahra M, Lyonnet D. Tubular ectasia of the rete testis: a potential pitfall in scrotal imaging. *Eur Radiol* 1999; 9:1862-8
- Langer JE, Ramchandani P, Siegelman ES, Banner MP. Epidermoid cysts of the testicle: monographic and MR imaging features. *AJR* 1999; 173(5):1295-9
- Yeung CK, Tam YH, Chan YL, Lee KH, Metreweli C. A new management algorithm for impalpable undescended testis with gadolinium enhanced magnetic resonance angiography. *J Urol* 1999; 162(3 Pt 2):998-1002
- Serra AD, Hricak H, Coakley FV, Kim B, Dudley A, Morey A, Tschumper B, Carroll PR. Inconclusive clinical and ultrasound evaluation of the scrotum: impact of magnetic resonance imaging on patient management and cost. *Urology* 1998;51(6):1018-21
- Lam WW, Tam PK, Ai VH, Chan KL, Cheng W, Chan FL, Leong L. Gadolinium-infusion magnetic resonance angiogram: a new, noninvasive, and accurate method of preoperative localization of impalpable undescended testes. *J Pediatr Surg* 1998;33(i):123-6
- Mattrey RF: MRI of the Scrotum and Testes, Chapter 47, In *Clinical Magnetic Resonance Imaging*, 2nd ed. Edleman et al, editors. W. B. Saunders Company, Philadelphia PA 1995.
- Tartar VM, Trambert MA, Balsara ZN, Mattrey RF: Tubular ectasia of the testicle: Sonographic and MR imaging appearance. *AJR* 1993; 160:539-542.
- Trambert MA, Mattrey RF, Levine D, Berthoty DP: Subacute Scrotal Pain: Evaluation of Torsion versus Epididymitis with MR Imaging. *Radiology*. 1990; 175:53-56.
- Johnson JO, Mattrey RF, Phillipson J: Differentiation of seminomatous from non-seminomatous testicular tumors with MR imaging. *AJR* 1990; 154:539-543.

- Baker LL, Hajek PC, Burtchard TK, Dicapua L, Leopold GR, Hesselink JR, Mattrey RM: Magnetic Resonance Imaging of the Scrotum: Normal Anatomy. *Radiology* 1987; 163:89-92.
- Baker LL, Hajek PC, Burkhard TK, Dicapua L, Landa HM, Leopold GR, Hesselink JR, Mattrey RM: Magnetic Resonance Imaging of the Scrotum: Pathologic conditions. *Radiology* 1987; 163:93-98.