

## Chapter 2

# Tissue Relaxation

### Nuclear Magnetic Resonance

At this point, we have referred to MR in two different ways: nuclear magnetic resonance (NMR) and magnetic resonance imaging (MRI). NMR is the analytical technique of exciting and obtaining MR signal from an entire sample, breaking the measured signal into its individual frequency components to identify different molecules containing the nucleus of interest, but not separating the signal by location within the sample. In contrast, MRI is the technique of separating a sample or region of tissue into individual volume elements (voxels) and producing images based on the total signal from the nucleus of interest in each voxel. The techniques of dividing tissue into voxels to produce MR images will be discussed in Chapter 3. This chapter describes tissue relaxation times that provide the contrast seen in MR images. Relaxation times are strictly MR-based parameters that describe the re-growth of longitudinal magnetization (T1) and loss of transverse magnetization (T2) after a radiofrequency (RF) pulse flips magnetization out of alignment with the externally applied static magnetic field,  $B_0$ .

In 1952, Bloch and Purcell were awarded the Nobel Prize in physics for independently demonstrating that NMR could be performed on liquids and solids.<sup>1,2</sup> Based on their work, equipment to perform quantitative NMR was developed and refined. An NMR spectrometer consists of a strong magnet, a radiofrequency (RF) transmitter coil tuned to the resonant frequency of the nucleus of interest, and a RF receiver coil tuned to the same frequency. NMR spectrometers are not designed to separate the sample into individual voxels but simply to measure the collective signal from a single sample. NMR systems require a highly homogeneous magnetic field over the entire sample so that all nuclei of a particular type in the sample have the same resonant frequency except for inherent small differences in resonant frequencies caused by slightly different nuclear shielding occurring due to different molecular structure.

For example, hydrogen nuclei in water resonate at a Larmor frequency that is higher by 3.4 parts per million (ppm) than hydrogen nuclei in fat. This is due to the slightly different molecular environment of the hydrogen nuclei in  $H_2O$  compared to that in  $CH_2$ . At 1.5 T, 3.4 ppm amounts to a frequency shift of 214 Hz in resonant

frequency. This is quite small compared to the differences in resonant frequency of different nuclei, shown in Table 1.2, which are MHz or tens of MHz. The nucleus with resonant frequency closest to that of hydrogen is fluorine-19, which has a gyromagnetic ratio of 40.4 MHz/T. At 1.5 T, F-19 resonates at a frequency of 60.6 Megahertz ( $1.5 \text{ T} \times 40.4 \text{ MHz/T}$ ) compared to a resonant frequency of 63.9 MHz for hydrogen, a difference of 3.3 MHz. The frequency shift between hydrogen in fat and hydrogen in water of 214 Hz is extremely small compared to the difference of 3,300,000 Hz between resonant frequencies of hydrogen and fluorine.

Most NMR spectrometers have small bore sizes that accommodate only small samples, because it is easier and less expensive to build a magnet with a uniform magnetic field over a small volume. Thus, typical spectrometer bore sizes are 10 cm or less. NMR spectrometers usually lack the magnetic gradients necessary to separate the sample into individual voxels.

In addition to using NMR as analytic tools to determine the chemical constituents of materials, by the early 1970s, researchers were beginning to measure NMR relaxation parameters of excised tissue samples. In so doing, they learned that tissue relaxation times differ between normal and diseased tissues. In 1971, Damadian demonstrated that the MR relaxation times, specifically the T1/T2 ratios of tissues, could be used to distinguish cancers from normal tissues.<sup>3</sup>

To a physicist, one of the remarkable aspects of MRI is that the signal coming from a hydrogen nucleus, which has a size of about  $10^{-15}$  meters (m), is sensitive to macroscopic disease in tissue. Most nuclear phenomena tell us only about the nucleus itself or, occasionally, about atomic structure, but are shielded from differences in tissues that occur at the size scale of macromolecules within cells. The reason that the magnetic properties of hydrogen nuclei can tell us something about disease is that the relaxation times T1 and T2 of hydrogen nuclei depend on their macromolecular environments. Therefore, while the magnetic dipole moments of hydrogen nuclei are the probes, these probes are sensitive to differences in their macromolecular environments through their relaxation times. Thus, signals coming from hydrogen nuclei are affected by phenomena that occur on the much larger size scale, that of macromolecules within cells, which are of submicron to micron sizes ( $10^{-8}$  to  $10^{-6}$  meters). Moreover, while the quantity of hydrogen within soft tissues might vary up to a few percent, T1 and T2 in different soft tissues can vary by up to 100%, making relaxation times more sensitive sources of contrast than hydrogen density alone.<sup>4</sup> This is one of the underlying reasons that MRI is exquisitely sensitive to disease.

The NMR experiment described at the end of Chapter 1 indicated that the maximum measurable MR signal occurs just after tissue magnetization is rotated into the transverse plane, perpendicular to  $B_0$ , by a  $90^\circ$  RF pulse. Two separate phenomena take place after a  $90^\circ$  pulse is applied. One phenomenon is the recovery of the longitudinal magnetization, described by the time constant T1 and called spin-lattice relaxation, T1 relaxation, or T1-recovery. The other phenomenon is the decay of transverse magnetization, the magnetization that is flipped into the transverse plane, which is described by the time constant T2 or T2\* and is called spin-spin relaxation or T2-decay. We will describe each of these phenomena separately.

## T1 Relaxation

T1-relaxation or T1-recovery describes the recovery of longitudinal magnetization along the direction of the static magnetic field,  $B_0$ , just after applying an RF pulse.<sup>4</sup> Recall that there are two energy states the hydrogen nucleus can occupy in the presence of an externally applied magnetic field: up, the lower energy state where the magnetic dipole moment points along  $B_0$ , or down, the higher energy state where the magnetic dipole points opposite  $B_0$ . Thinking of tissue magnetization as a collection of hydrogen dipoles, after a  $90^\circ$  RF pulse there is an equal population of hydrogen dipoles in the lower energy and higher energy states. T1-relaxation describes the recovery of longitudinal magnetization due to thermal interactions between excited, higher energy hydrogen nuclei (spins) and nearby, large macromolecules within the sample (the lattice). These interactions decrease the number of higher energy state (down-oriented) dipoles and increase the number of lower energy (up-oriented) hydrogen nuclear dipoles. As these energy transitions take place, the strength of tissue magnetization pointing along  $B_0$  increases. Because T1 recovery involves interactions between spins and the larger surrounding lattice, it is sometimes referred to as “spin-lattice” relaxation.

This phenomenon of recovery of longitudinal magnetization is described by the relaxation time T1. The thermal interactions that cause T1-recovery occur primarily when tissues contain large macromolecules that have appropriate energy states to absorb the exact amount of energy to allow hydrogen nuclei to go from their excited, higher-energy state to their lower-energy state. The amount of energy released by the hydrogen nucleus equals the energy absorbed by the macromolecule.

As described in Chapter 1, the amount of energy released by the hydrogen dipole undergoing this transition from higher to lower energy states is determined by the magnetic field strength and by the nuclear type undergoing this transition, which in MRI is hydrogen. The required energy transfer is given by the Larmor equation (Eq (1.3) or Eq. (1.4)):

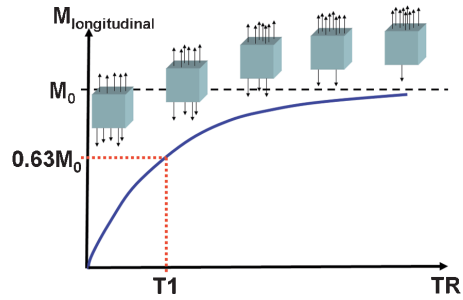
$$\Delta E = h\nu = h\omega_0 / (2\pi) = \mu_p B_0, \quad (2.1)$$

where  $h$  is Planck’s constant,  $\nu$  is the frequency in units of cycles per second ( $1/s$  or  $s^{-1}$ ),  $\omega_0$  is the Larmor frequency in units of radians per second,  $\mu_p$  is the magnetic moment of the proton, and  $B_0$  is the magnetic field strength in which the proton resides.

The rate at which this spin-lattice energy transfer takes place is one minus an exponential, reflecting the fact that the longitudinal magnetization starts out as zero just after a  $90^\circ$  pulse and recovers quickly at first, since there are a large number of higher energy dipoles. As time progresses and more dipoles flip to their lower energy state along  $B_0$ , recovery slows because there are fewer hydrogen dipoles available in the excited, higher energy state to make the energy transition (Figure 2.1). The equation that describes the strength of longitudinal magnetization,  $M_z(t)$ , at a time  $t$  after the application of a  $90^\circ$  pulse is:

$$M_z(t) = M_0(1 - e^{-t/T1}), \quad (2.2)$$

**Figure 2.1** Illustration of the population change as T1-relaxation occurs in a voxel of tissue. Over time, fewer and fewer hydrogen dipoles are in the higher-energy state (down) and the number of energy transitions from higher to lower energy (up) states is fewer, slowing the rate of recovery of longitudinal magnetization. T1 is defined as the time after a 90° pulse needed for the longitudinal magnetization to recover to  $(1-e^{-1})$  or 63% of  $M_0$ , the longitudinal magnetization achieved with infinite TR.



where  $M_0$  is the maximum longitudinal magnetization occurring in the sample at that magnetic field strength and  $t$  is the time since the 90° pulse. T1 is defined as the time it takes after a 90° pulse for the longitudinal magnetization to recover to  $M_0(1-e^{-1}) = M_0(1-(1/(2.713))) = 0.63M_0$ ; that is, to 63% of its maximum possible strength. Mathematically, it takes an infinite amount of time for the longitudinal magnetization to fully recover to  $M_0$ , the magnetization it had before the 90° pulse was applied. In practical terms, however, the longitudinal magnetization is 95% recovered along  $B_0$  after a time interval equal to  $3T1$ , 99% recovered after  $5T1$ , and 99.9% recovered after  $7T1$ .

Tissues with more macromolecules of the correct size to enable these thermal interactions have shorter T1 values, indicating that the energy exchange between hydrogen nuclei and macromolecules occurs more rapidly. Tissues with very dilute concentrations of macromolecules, such as cerebrospinal and cystic fluids, have long T1-values, on the order of several seconds, because energy transfer occurs more slowly. In the breast, T1 values are shortest for fat (about 250 ms at 1.5 T), intermediate for fibroglandular tissues (about 700 ms at 1.5 T), higher for most lesions, including cancers (800 ms to 1 second at 1.5 T), and highest for non-bloody cystic fluids (about 3 seconds at 1.5 T). The reason that T1 is higher for most breast lesions, including cancers, than for normal fibroglandular tissues is that lesions tend to have higher water concentrations, and therefore fewer macromolecules per unit volume, than normal breast tissues. The exceptions to this rule are lesions with high fat content, such as lipomas, or lesions with a high fibrous content, both of which have shorter T1 values than normal fibroglandular tissues.

The primary effect of administering gadolinium chelates, such as Gd-DTPA, is to shorten the T1 relaxation times of hydrogen nuclei.<sup>5</sup> Gd-chelates, like other macromolecules, act as energy sponges, absorbing the energy needed for the hydrogen nuclei to transition from their excited to unexcited states. Hence, when Gd-chelate molecules are present in adequate numbers, T1 is shortened. Since Gd-chelates are selectively taken up by lesions, especially cancerous lesions that have recruited more vessels and capillaries to support tumor growth, the T1 relaxation times of lesions are shortened from 800 to 1000 ms to 200 to 400 ms on the first pass of Gd-chelates, making the recovery of longitudinal magnetization faster in enhancing

lesions than in all other breast tissues except fat. The fact that enhancing lesions have similar T1-values to fat is why enhancing lesions often are isointense with fat on non-fat-suppressed T1-weighted images.

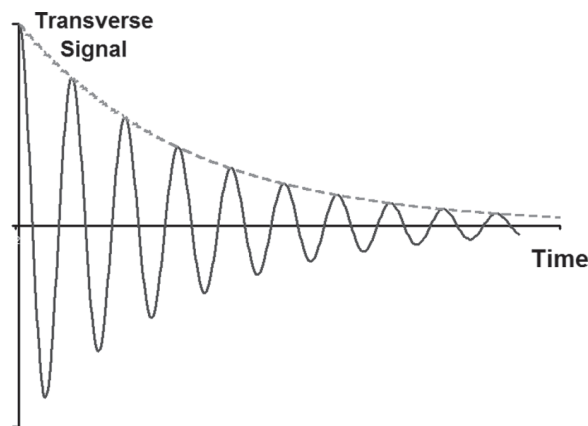
## T2 Relaxation

If the transverse magnetization is measured just after a  $90^\circ$  pulse, the measured signal oscillates at the Larmor frequency and decreases quickly over time, as shown in Figure 2.2. The main reason transverse magnetization is lost is that the magnetic dipoles of hydrogen nuclei begin to dephase because different hydrogen nuclei have subtly different magnetic field environments and therefore precess at slightly different rates (Figure 2.3). This difference in precessional frequencies means that the transverse magnetization decreases in magnitude over time. The dephasing of the measurable MR signal, the transverse magnetization, is described by T2 or T2\*, depending on how the transverse signal is formed before being measured.

Without distinguishing between T2 and T2\* yet, T2-type decay describes the exponential loss of transverse magnetization immediately after a  $90^\circ$  pulse. T2 is the parameter that describes how quickly the magnitude of the transverse magnetization decreases over time. The shorter T2, the more rapidly transverse magnetization decreases. The transverse magnetization,  $M_{xy}(t)$ , at a time  $t$  after a  $90^\circ$  RF pulse is described by the equation:

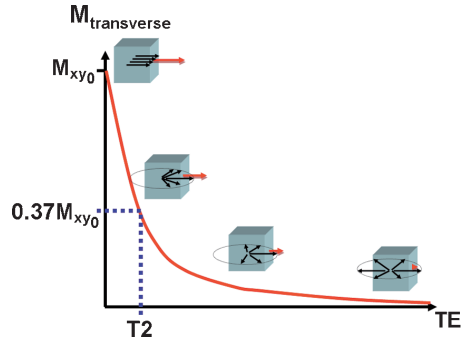
$$M_{xy}(t) = M_{xy} e^{-t/T2}, \quad (2.3)$$

where  $M_{xy}$  is the transverse magnetization immediately after a  $90^\circ$  pulse, at  $t=0$ . The magnitude of measured transverse magnetization is observed to have an exponential rate of decrease (Figure 2.3). The parameter T2 in the above equation is



**Figure 2.2** Measured transverse magnetization oscillates at the Larmor frequency and quickly decreases in magnitude due to T2 or T2\* decay.

**Figure 2.3** The dephasing of hydrogen dipoles flipped into the transverse plane over time and is responsible for T2 (or T2\*) decay. T2 (or T2\*) is defined as the time needed for the transverse magnetization to decrease to 1/e (or 37%) of the signal strength in the transverse plane just after a 90° pulse.



defined as the time it takes for the signal to decrease to 1/e or to 37% of the original signal strength it had just after the 90° pulse. Hence, tissues with shorter T2-values have a more rapid loss of transverse magnetization. In the breast, fat and normal fibroglandular tissues have the shortest T2 values (60 to 80 ms). Most breast lesions, including cancers, have slightly longer T2 values (80 to 100 ms), and cystic fluids have the longest T2-values (several hundred ms).

Mathematically, it takes an infinite amount of time for transverse magnetization to completely disappear, but after 3T2 the transverse magnetization is at 5%, and after 5T2 it is at 1% of its original strength.

For a given tissue, T2 is always shorter than T1 because the rate at which transverse magnetization decreases is faster than the rate at which longitudinal magnetization recovers along  $B_0$ . This is because T1-recovery is only one of several reasons that the measurable transverse signal decreases after a 90° pulse. If T1-recovery were the only reason for signal loss, then T2 would equal T1 and only one decay parameter would be needed in NMR or MRI. Because there are other, more dominant sources of transverse magnetization loss, the transverse signal decreases more rapidly than the longitudinal magnetization recovers. Hence, two separate NMR parameters are needed, T1 and T2, with T2 shorter than T1, usually by a factor of 5 to 10.

The dephasing of magnetic dipoles making up the tissue magnetization from a sample or voxel is similar to a grade-school ballet ensemble performing Swan Lake. The dipoles (dancers) start out pointing in the same direction as they precess (turn) in unison, but as more rotations take place, they start pointing in different directions due to their slightly different rates of precession (turning). In Swan Lake, the ensemble turns once every few seconds, but it takes only a few turns for inexperienced dancers to point in different directions. In hydrogen nuclei at 1.5 T, magnetic dipoles precess 64,000 times every millisecond, so it takes only subtly different magnetic field environments for the dipoles to point in slightly different directions after a few milliseconds. This dephasing decreases the measurable amount of transverse magnetization and is responsible for T2-decay.

In T2-decay, one hydrogen dipole (a “spin”) creates a non-uniform magnetic environment for other hydrogen dipoles (other “spins”) nearby. Therefore, these subtle magnetic interactions that are the main cause of T2-decay are called “spin-spin” interactions. This type of dephasing is referred to as “true T2 dephasing,”

because it results from the inherent molecular environment, which changes rapidly in time, and cannot be reversed by gradient or RF pulse manipulations. These true T2 dephasing effects are excellent indicators of disease. It is these dephasing effects that MRI depends on to distinguish one tissue from another in T2-weighted imaging.

The role that macromolecules play in T2 relaxation is different than the role they play in T1-recovery. In pure water, where there are few or no macromolecules, there are plenty of hydrogen dipoles to affect the magnetic environments of other hydrogen dipoles, but the rate at which water molecules are moving is so rapid that each hydrogen dipole “sees” or experiences a relatively uniform magnetic environment. Thus, T2 is relatively long in pure water. As more macromolecules are added to water, the rate of motion of water molecules is slowed. This slowing means that the average amount of time that a hydrogen nucleus spends in contact with other molecules is longer. This increases the non-uniformity in the magnetic environment experienced by each hydrogen dipole, making dephasing occur more rapidly and thus making T2 shorter.

This effect is responsible for the subtly different magnetic environments of hydrogen nuclear dipoles and the resulting T2 differences between normal and diseased tissues. Diseased tissues typically are more edematous than normal tissues and therefore have lower concentrations of macromolecules. Therefore, T2 in diseased tissues is typically longer because hydrogen nuclei move more rapidly and the magnetic environment seen by each hydrogen nucleus is more uniform.

Gadolinium-based paramagnetic contrast agents shorten T2 as well as T1, since adding Gd-chelates has a similar effect to adding macromolecules to tissues. The primary reason that T1-weighted imaging, rather than T2-weighted imaging, is used with Gd-based contrast agents is that T1 is much longer than T2 in unenhanced tissues; as a result, the fractional change in T1 due to contrast agent uptake is greater than the fractional change in T2.<sup>5</sup> Thus, T1-weighted imaging shows a bigger effect of the contrast agent than T2-weighted imaging. In addition, we will see later that T1-weighted imaging can be done more rapidly than T2-weighted imaging. Finally, we will also see later that Gd-chelates act as a positive contrast agent on T1, making lesions that take up Gd-chelates brighter, but act as a negative contrast agent on T2. The convention in MRI is that higher signal is depicted as brighter in the MR image. Gd-chelates shorten T1 and T2. Shortening T1 causes signal to be higher on T1-weighted images, making lesions taking up gadolinium to be brighter. Shortening T2 causes signal to be lower on T2-weighted images, making lesions that take up gadolinium darker. There is a viewer preference for searching for bright enhancing areas rather than darker, suppressed areas on a heterogeneous background such as the breast, so positive contrast is preferred.

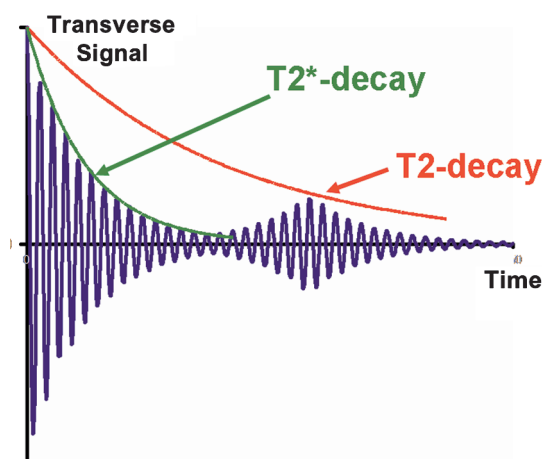
## **Distinguishing T2 and T2\***

There is another cause of transverse magnetization dephasing beyond true T2-effects: namely, non-uniform magnetic fields across the sample or voxel of tissue.<sup>6</sup> These magnetic field inhomogeneities are usually caused by external factors and

thus are not useful indicators of disease. Magnetic field inhomogeneities may be due to a non-uniform primary magnetic field ( $B_0$ ), intentional applications of magnetic gradients that are needed to resolve the sample into voxels, and magnetic non-uniformities due to the presence of metallic objects or differences in magnetic susceptibility (the ability of tissues to maintain a magnetic field) in the tissue itself.  $T2^*$  includes all of these causes of transverse magnetization dephasing.  $T2^*$  is the dephasing parameter that applies to “free-induction decay,” the decay occurring when the transverse magnetization is measured just after a  $90^\circ$  pulse, without any attempt to “rephase” it, as shown in Figure 2.2. Thus, the simple NMR experiment described at the end of Chapter 1 is a free-induction decay experiment, and  $T2^*$  governs the decay of the magnitude of measured transverse magnetization in that experiment according to Equation 2.3, with  $T2^*$  replacing  $T2$ .  $T2^*$  is shorter than  $T2$  because it includes magnetic field inhomogeneities as a way to decrease transverse magnetization:

$$1/T2^* = 1/T2 + \gamma\pi\Delta B_0 \quad (2.4)$$

where  $\gamma$  is the gyromagnetic ratio and  $\Delta B_0$  is the magnetic field inhomogeneity across a voxel, including all possible causes. In Eq. 2.4, since  $1/T2^*$  is made larger by the additional term due to magnetic field inhomogeneities ( $\Delta B_0$ ),  $T2^*$  is smaller than  $T2$ . In extremely homogeneous magnetic fields,  $T2^*$  values are approximately equal to  $T2$  values. In voxels with magnetic inhomogeneities due to either non-uniform magnetic fields or magnetic susceptibility inhomogeneities,  $T2^*$  will be shorter than  $T2$ . Even being in a different location in the MR scanner, away from isocenter, can lower  $T2^*$ , but not  $T2$ . Figure 2.4 illustrates the difference between  $T2$  and  $T2^*$  decay.

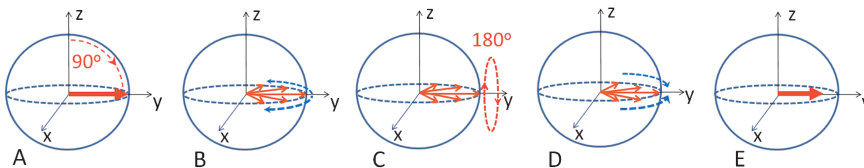
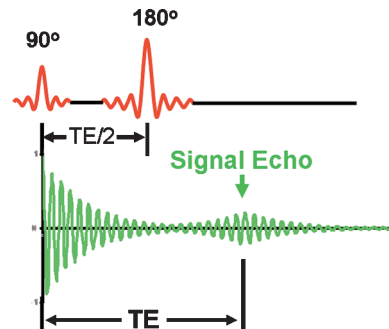


**Figure 2.4** Illustration of the difference between  $T2^*$  and  $T2$ .  $T2$  describes the decay between initial transverse magnetization and the peak magnetization formed by a spin-echo.  $T2^*$  describes the decay due to true  $T2$  relaxation plus magnetic field non-uniformities across the sample or voxel.



T2 describes the dephasing of transverse magnetization when a spin-echo is formed. A spin-echo is formed by applying a  $180^\circ$  RF pulse halfway between the  $90^\circ$  pulse and peak signal measurement (Figure 2.5). Prior to the  $90^\circ$  pulse, some dipoles precess faster than others due to both irreversible effects (true T2 decay) and reversible effects (such as non-uniform magnetic fields across the sample or voxel). The effect of the  $180^\circ$  pulse is to exchange the orientation of the faster precessing hydrogen dipoles with that of slower precessing hydrogen dipoles. The  $180^\circ$  pulse flips the faster precessing dipoles into the position of slower precessing dipoles, and vice versa (Figure 2.6). The beauty of a spin-echo is that it eliminates almost all sources of T2 dephasing other than true T2 effects; that is, spin-echo pulse sequences eliminate reversible dephasing effects, such as static magnetic field non-uniformities, gradient non-uniformities, and magnetic susceptibility effects. Hence T2 is greater than T2\*, as shown in Figure 2.4. Thus, T2-weighted spin-echo imaging is more robust than free-induction decay in that it yields the greatest measured signal for a given delay time. T2-weighted spin-echo imaging is also more useful diagnostically because it focuses on true T2-decay effects that reflect inherent tissue differences rather than including system effects such as magnetic field, gradient, or magnetic susceptibility differences.

**Figure 2.5** Formation of a spin-echo by applying a  $180^\circ$  pulse halfway between the  $90^\circ$  pulse and signal measurement. The  $90^\circ$  pulse flips longitudinal magnetization into the transverse plane, where it can be measured. The application of a  $180^\circ$  pulse forms the signal echo and eliminates the dephasing effects of magnetic field uniformities.



**Figure 2.6** (A) After a  $90^\circ$  pulse, hydrogen magnetic dipoles are precessing in-phase. These diagrams are in the “rotating frame” where dipoles precessing at exactly the Larmor frequency are pointing along the y-axis. (B) After a short time, faster precessing dipoles have advanced in phase relative to more slowly precessing dipoles. (C) A  $180^\circ$  pulse is applied to the sample. This causes the magnetization vectors of faster precessing dipoles to be rotated to the orientation of slower dipoles, and vice versa. (D) Over the next brief interval, reversible effects are rephased. (E) An equal time after the  $180^\circ$  pulse, maximum signal is rephased in the transverse plane.

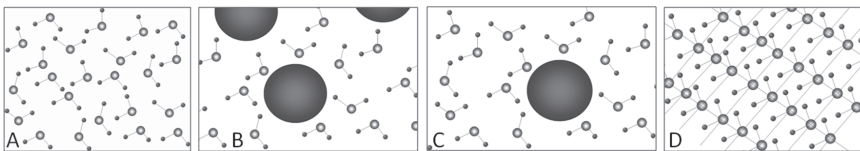
## The Physical Basis of Relaxation Times

This section summarizes the effects of tissue properties on T1 and T2 relaxation times. Pure water, CSF, or clear cystic fluids have the longest T1 and T2 values. T1 values in these fluids are long (a few seconds) because there are few macromolecules to absorb energy from excited hydrogen magnetic dipoles and allow them to transition to lower energy states (Figure 2.7A). T2 in these fluids are long, but not as long as T1, because the rapidly moving water molecules create a relatively uniform magnetic environment for all hydrogen nuclei, so there is slow dephasing of hydrogen magnetic dipoles after a 90° RF pulse.

Normal fibroglandular tissues have shorter T1 and T2 values than water or cystic fluid. T1 is shorter among normal breast tissues because there are sufficient numbers of macromolecules to absorb the energy of excited hydrogen nuclei and permit them to transition to the lower energy states (Figure 2.7B). T2 is shorter in fibroglandular tissues than in pure water, CSF, or cystic fluid because the higher concentration of macromolecules in normal breast tissue slows the motion of water molecules, causing them to experience greater magnetic field non-uniformities and therefore dephase more rapidly.

Breast cancers have T1 and T2 values somewhat higher than those of normal fibroglandular tissues because they are typically edematous, with lower concentrations of macromolecules than fibroglandular tissues (Figure 2.7C). Therefore, their T1 and T2 values are slightly longer than those of fibroglandular tissues but not nearly as long as CSF or cystic fluids.

Water molecules locked in a crystalline or lattice structure (Figure 2.7D) tend to have longer T1 values because the exchange of energy between hydrogen nuclei and macromolecules is limited, but shorter T2 values than in A-C because of the large magnetic field inhomogeneities maintained by the lattice structure.



**Figure 2.7** (A) Distilled water consisting of randomly organized water molecules. A moving picture would show individual water molecules moving so rapidly that each hydrogen dipole would see a uniform magnetic field environment, causing T1 and T2 to be very long. (B) Water with macromolecules typical of a normal cell. The macromolecules slow down water molecules in their vicinity, undergoing thermal collisions to take up energy from excited hydrogen dipoles, shortening T1, and providing magnetic field non-uniformities, which shortens T2. (C) Dilute macromolecules, typical of a cancer or benign lesion. Here, T1 and T2 are shortened by the mechanisms described in (B), but to a lesser extent due to the lower concentration of macromolecules. (D) In a solid or semi-solid lattice, water molecules are locked more rigidly in place within the lattice, making T1 longer due to fewer possible energy transfers from spins to the lattice, but T2 very short due to magnetic inhomogeneities caused by the lattice.

Fat has the shortest T1 values in the breast because of the presence of a sufficient number of macromolecules to enable the rapid transition of hydrogen nuclei in CH<sub>2</sub> from the excited higher energy state to the lower energy state. T2 values of hydrogen in fat are similar to, or slightly longer than, those in fibroglandular tissues primarily because the motion of hydrogen in CH<sub>2</sub> is similar to the motion of hydrogen in H<sub>2</sub>O in fibroglandular tissues, so T2 dephasing effects are similar.

The rule that cysts have longer T1 and T2 values is broken by bloody cysts. Blood, like Gd-chelates, is a paramagnetic agent, due to unpaired electrons in iron ions in hemoglobin. As a result, blood-filled cysts have shorter T1 and T2 values than normal cystic fluid and can even have shorter T1 and T2 values than normal breast tissues, making blood-filled cysts appear brighter than fibroglandular tissues on T1-weighted images and darker than fibroglandular tissues on T2-weighted images.

These basic concepts are responsible for the contrast observed in MRI of the breast. To understand how T1 and T2 contribute to image contrast, we need to describe how MR pulse sequences manifest T1 and T2 contrast, which is described in detail in Chapter 4 and beyond.

## Chapter Take-home Points

- Longitudinal and transverse relaxation times are determined largely by the macromolecular environment of hydrogen nuclei.
- The more macromolecules of the correct size, the shorter T1.
- The more slowly water molecules move and the longer they spend in the vicinity of larger molecules, the shorter T2 and T2\*.
- Diseased tissues tend to have longer T1 and T2 values, and higher spin-densities, than normal tissues.
- The uptake of Gd-chelates in tissues causes T1, and to a lesser extent T2, to shorten dramatically; this makes lesions with Gd-chelate uptake bright on T1-weighted sequences because tissues with shorter T1 values have higher signal.

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Breast MRI

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