Introduction. Diffusion refers to the random Brownian molecular motion. In a bulk liquid, water molecules are free to move and this is characterized by a large value of the diffusion constant, $D$. The motion of water molecules within cells may be restricted by the cell wall and other structures, resulting in a lower value of $D$; the same may be true of interstitial fluid in tissues if the cells are densely packed. Diffusion-Weighted Imaging (DWI) generates images based on the variation of $D$ [1].

Diffusion-Weighted MRI. DWI uses balanced pulses of magnetic field gradient, applied either side of a 180° pulse within a spinecho pulse sequence, as introduced by Stejskal and Tanner in 1965 [2]. For stationary spins, any phase accrued during the first gradient pulse is unwound during the second pulse. Spins in random motion, however, become dephased, leading to a diffusion-dependent loss of signal. The signal can be given by $S(b, TE) = M_0 \exp\left(-\frac{b}{2} T_e\right) \exp(-bD)$ where $b$ is a factor which is dependent on the square of gradient strength ($G^2$), its duration and spacing, known as the “$b$-value”; the stronger the value of $b$, the higher is the diffusion weighting [3]. $b$ values in the range 0–1000 s/mm² are typically used. In a DWI image with a large $b$-value, tissues containing freely-diffusing water molecules will appear dark (due to loss of signal), while tissues where diffusion is restricted will appear with greater intensity.

Conclusions. DWI is a standard part of the diagnostic armoury of clinical MRI, where one of its most important applications lies in the diagnosis of cancer. This lecture will describe the basic principles of DWI, with particular emphasis on its use in oncology [4].

References


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[1003] Diffusion MRI: Sequence optimisation in oncology
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The use of alpha-particle emitting radionuclides for therapy of primarily disseminated cancers has been proposed for at least half a century. The last few years, this modality has been introduced into clinical practice. Today, Xofigo (Ra-223) is commercially available and clinical trials with the alpha-emitters At-211, Bi-213, Th-227, Pb-212 and Ac-225 have been initiated. With the approval of an alpha-emitting radio-pharmaceutical, and several others in clinical pipeline, improvements in clinical alpha-particle dosimetry are urgently needed.

This talk will discuss how dosimetry can and should guide optimizations of various alpha-particle therapies and thereby also fulfill the requirements set up by new regulations for patient-specific dosimetry. This includes the range from measuring whole-body retention using simple probes to microdosimetry on individual cells imaged by quantitative alpha-cameras.

As an example, retrospective dosimetry on patients enrolled in our clinical trial on intraperitoneally administered $^{211}$At-MX35 F (ab')2 for therapy of disseminated ovarian cancer will be presented. That study included clinical biodistribution data from blood and intraperitoneal fluid sampling, urine collection, and scintigraphy from twelve patients. Mean absorbed doses to bone marrow, thyroid, urine bladder and peritoneum were estimated. The uptake in liver, heart, lungs, kidneys and breasts was quantified using SPECT images.

Dosimetry can and should be used to predict therapeutic effect, but also for estimating possible risks. Such benefit/risk estimates can then be used to further optimize the various alpha-particle therapies that are now being introduced in the clinic.

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