The value of b required to avoid T2 shine-through from old lacunar infarcts in diffusion-weighted imaging

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Abstract
Multiple small infarcts of different ages are common in small-vessel disease. Diffusion-weighted imaging (DWI) is a powerful method for discriminating new from chronic lesions. This can be done on the diffusion-weighted images provided that b is sufficiently high. Our purpose was to determine that critical value of b. We reviewed DWI from a previous study of acute, mainly lacunar strokes, and selected 18 old lacunar infarcts, well defined on uncoded images with b 0 s/m² (i.e., T2-weighted images) but invisible on DWI with b 1200 × 10⁶ s/m². We used a 1.5 tesla imager and single-shot echo-planar technique. We had seven separate acquisitions with echo time 123 ms and b in steps between 0 and 1200 × 10⁶ s/m². Two neuroradiologists blinded to the selection of lesions carried out two different lesion-detection procedures, thereby testing each lesion four times, giving a total of 72 tests of b values. The results were consistent, indicating a level for detection of 800 × 10⁶ s/m² in two tests, 400–600 × 10⁶ s/m² in 65 tests and at lower values in the remainder. For imagers up to 1.5 tesla, at long repetition times and an echo time up to 120 ms T2-shine through of old lacunar infarcts can be avoided using b of 1000 × 10⁶ s/m².

Keywords
Lacunar infarcts · Diffusion-weighted imaging

Introduction
Diffusion weighted MRI (DWI) is a powerful way of detecting acute infarcts [1, 2, 3, 4, 5, 6, 7, 8]. Lesions of lacunar type, not involving grey matter can be difficult to show with conventional T2-weighted images, sometimes for more than 24 h after ischaemic event [9, 10, 11]; the sensitivity is the same as that of CT [12]. In small-vessel disease multiple small ischaemic lesions of different ages are common. Identification of a new ischaemic lesion among other pre-existing foci may be desirable. DWI is therefore of interest for differential diagnosis, even beyond the initial hours after the onset of a stroke [13, 14].

A new ischaemic lesion gives higher signal than the surrounding tissue on DWI. This disappears with ageing of the lesion. Time courses for these changes have been described [15, 16, 17]. The factors influencing signal intensity in spin-echo (SE) DWI are given by the equation [18]:

\[ S_{DWI} = S_{T2} \cdot e^{-b \cdot D} \]  

(1)

where \( D \) is the apparent diffusion coefficient (ADC, m²/s), \( S_{T2} \) the signal in the corresponding T2-weighted image and b the diffusion-sensitising coefficient (s/m²) of the DWI pulse sequence. After an almost instant decrease in the ischaemic tissue (the basis for early visibility on DWI) the ADC will rise to a level substantially above that of normal tissue in the chronic stage. Burdette et al. [19] demonstrated this process in the acute and subacute stages, the first 7 days after onset of a cerebral infarct. Given the high signal of a chronic infarct on T2-weighted images it is obvious, from equation 1 that
the value of $b$ is crucial in ensuring that the T2-dependent signal component of the ischaemic lesion does not shine through (“T2-shine through”) when DWI is used to discriminate between new and old lesions.

An ADC map can be calculated pixel-by-pixel from two or more acquisitions with different values of $b$, using equation 1. Automated ADC calculation is an option on modern imagers. The triad of T2-weighted images, DWI and an ADC map provides a complete basis for interpretation of ischaemic lesions, enabling full interpretation even if $b$ is not high enough to suppress the signal from chronic infarcts on DWI. A new lesion with a low ADC can usually be differentiated from chronic lesions with a high ADC on the ADC map. However, the latter is a synthesis of two or more separate acquisitions. In small infarcts, the slightest movement of the patient between acquisitions can obscure the lesion on the map. DWI does not suffer from this drawback. Acute and subacute lesions can be separated from chronic lesions in a quick, safe and convenient way directly on DWI with a sufficiently high $b$.

In a previous study [15] we found that with $b = 1200 \times 10^3 \text{ m}^2/\text{s}$ the high signal of an ischaemic lesion virtually always disappeared in the chronic stage. However, a high $b$ requires high-performance gradients. A high $b$ also conflicts with image quality by decreasing the signal-to-noise ratio and increasing geometrical image distortion caused by the diffusion-sensitising gradients (when echo-planar techniques are used). Image distortion is especially troublesome when integrating acquisitions with different diffusion-encoding directions. It is therefore important to determine how high $b$ has to be for reliable suppression of the T2-dependent high signal from of old lacunar infarcts. Our aim was to establish this critical level of $b$.

We reviewed the DWI from a previous study [9] of acute stroke in 27 patients, approved by the local ethic committee. We selected 18 chronic lacunar infarcts (lesions 1–18) in 11 patients (seven men, four women aged 52–77 years, up to three lesions per patient). The lesions had to be well-defined on un coded ($b = 10^3 \text{ m}^2/\text{s}$) images (i.e., the T2-weighted echo-planar image) but not visible on DWI with $b = 1200 \times 10^3 \text{ m}^2/\text{s}$. They were in white and deep grey matter, like the lacunar infarcts found at autopsy by Fischer [20] in 1042 adult brains. We therefore thought they adequately represented old lacunar infarcts.

A $b$-value series for a typical lesion is shown in Fig.1 (lesion 11). Only lesion 8 was in an area with interfering anisotropy effects (signal elevation at higher values of $b$ due to unfavourable orientation of fibre tracts); it lay at the edge of the affected area (Fig.2). Until anisotropy effects became prominent, with higher values of $b$, the lesion followed a pattern similar to Fig.1. Despite some influence of anisotropy at high $b$, we decided not to exclude this lesion.

Four of the lesions had the signal properties of a cavity on the PD and T2-weighted FSE images, the fluid within the cavity fluid having the signal properties of the cerebrospinal fluid. All these cavities showed a surrounding rim of high signal on PD images, similar to that of the lesions without cavitation.

Each lesion, examined with seven values of $b$ (seven images in exactly the same slice position), was photographed on a single sheet of film, which was cut into individual “image cards” (seven separate cards per lesion, one for each value of $b$). Three identical sets of cards were produced for each lesion. In addition to the well-defined lesion, the image in some cases also contained subtle lesions not considered appropriate for analysis.

The material was interpreted by two experienced neuroradiologists (SH and PCS) blinded to the selection of lesions. Each performed two different procedures, in each of which the interpreter was instructed to indicate any high signal which could represent an ischaemic lesion, despite the fact that he or she might thereby also indicate the subtle additional lesions and, in some cases, the new lesion when this lay in the imaging-plane.

In the first procedure, “the random-order mode”, each image card was given a random identification code which was impossible to associate with other cards of the same or other lesions. One set of cards from each lesion was assembled into a stack containing 7 × 18 (126) cards. The bundle was thoroughly mixed to obtain a completely random order. Each interpreter received a separate stack and was told to examine the cards one-by-one in the order given. Possible lesions were marked directly on the images. The cards were then sorted lesion-by-lesion. For each lesion and each $b$ a note was made in a table as to whether the lesion had been correctly identified. Lesions identified but not selected for the study were ignored.

The second procedure, “the serial mode”, was carried out 2 months later. The third set of image cards was sorted lesion-by-lesion. For each lesion the images were sorted in the order descending $b$. A supervisor (BG) presented the images to each interpreter lesion-by-lesion. The cards were presented starting with the highest $b$ ($1200 \times 10^3 \text{ m}^2/\text{s}$). If the interpreter could not point out the lesion, the next lower value of $b$ was presented, and this was repeated until the interpreter was able to point out the lesion. The $b$ at which the lesion was properly identified was noted as the detection level.

**Materials and methods**

All examinations were performed using a 1.5 tesla imager, maximum gradient strength 25 mT/m, minimum rise time 300 us, and the standard circularly polarised head coil. A deflatable vacuum pillow was used for fixing the head. We used SE echo-planar technique. The diffusion-encoding gradients were applied in the slice-selection direction only, with $b$ of 0, 270, 400, 600, 800, 1000 and $1200 \times 10^3 \text{ m}^2/\text{s}$. To minimize the influence of motion we used ECG triggering with the excitation pulse 350 ms after the peak of the R-wave. One slice was obtained following every fourth beat to allow complete relaxation before each excitation. The acquisitions with one $b$ were completed before the next value was applied. We usually obtained 11 slices covering the entire brain, with the following parameters: TE 123 ms, field of view (FOV) 250 × 250 mm² (320 × 320 mm² in some patients), matrix 128 × 200, slice thickness 8 mm, gap 0.8 mm. Conventional proton density (PD) and T2-weighted fast SE (FSE) images were also obtained: TR 3710 TE 22–90 ms, echo-train length 5, matrix 220 × 256, FOV 173 × 230 mm, slice thickness 5 mm, gap 1 mm.