**Field-Cycling Magnetic Resonance Imaging for identifying Minor Ischemic Stroke below 0.2 tesla.**

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<td>RAD-23-2972.R2</td>
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<tr>
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<td>Technical Development</td>
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Field-Cycling Magnetic Resonance Imaging for identifying Minor Ischemic Stroke below 0.2 tesla.

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Manuscript type
Technical development

Summary Statement
Field Cycling Imaging, a newly developed technology which measures changes in T₁ relaxation time-constant of tissues over a range of magnetic field strengths, can identify sub-acute ischemic stroke at field strengths between 0.2 and 200mT.

Key Results
In this prospective study of 9 participants with sub-acute ischemic stroke, infarct regions were identified using endogenous T₁ contrast mechanisms at field strengths between 200mT and 0.2mT with inter-rater agreement of 85.7%, Cohen’s kappa 0.69.

T₁ relaxation time-constants at ultra-low fields between 0.2T and 0.2mT were higher in infarct regions than in contralateral unaffected brain tissue (mean range 612 – 95ms vs 470 – 36ms; P range .018 – .109).

The contrast ratio between infarct and contralateral brain region increased as magnetic field strength decreased (r(26) = -0.68, P <.001).
Abstract

Background: Field Cycling Imaging (FCI) is a new technology developed at the University of Aberdeen which measures change in T<sub>1</sub> relaxation time-constant of tissues over a range of low magnetic field strengths (0.2mT – 200mT) by rapidly switching between different fields during the pulse sequence. This provides new sources of contrast, including some invisible to clinical MRI scanners, and may be a useful alternative imaging modality for stroke.

Purpose: To test whether a prototype whole-body FCI scanner can identify infarct regions in patients with subacute ischemic stroke.

Methods: This prospective study screened consecutive adult patients admitted to a single center stroke unit between February 2018 to March 2020 and April 2021 to December 2021. Included participants with confirmed ischemic stroke underwent FCI one to six days after ictus. FCI images were obtained at four to six evolution fields between 0.2mT and 0.2T with five evolution times from 5 – 546ms. T<sub>1</sub> maps were generated. Wilcoxon signed-rank test was used to compare infarct region and contralateral unaffected brain, and Spearman’s rank correlation to examine associations between infarct-to-contralateral tissue contrast ratio and field strengths. Two independent readers blinded to clinical images rated the FCI images.

Results: Nine participants (mean age [SD] 62.3 ± 15.5; 9 males) successfully completed FCI. FCI images below 0.2 T exhibited hyper-intense T<sub>1</sub> regions corresponding to the infarct region identified on baseline imaging, visually confirmed with 85.7% intrarater agreement (Cohen’s kappa 0.69). Infarct-to-contralateral tissue contrast ratio increased as magnetic field decreased between 0.2T and 0.2mT (r(26) = -0.68, P <.001). T<sub>1</sub> dispersion slopes differed between infarct and unaffected tissues (median [IQR]: 0.23 [0.18 – 0.37] vs 0.35 [0.27 – 0.43]; P = .03).

Conclusion: Whole brain FCI imaging can be used to identify subacute ischemic stroke by T<sub>1</sub> relaxation mechanisms at fields down to 0.2mT.
Abbreviations

FCI: Field Cycling Imaging

NMRD: Nuclear Magnetic Resonance Dispersion

FFC-NMR: Fast Field-Cycling Nuclear Magnetic Resonance

RF: RadioFrequency

$%\Delta T_1$: percentage difference in $T_1$
**Introduction**

The need for rapid informative imaging to direct stroke patients to endovascular centers, and increasing follow-up imaging has led to interest in modalities which do not have safety issues related to radiation dose or magnet strength.\(^1\)\(^-\)\(^3\) One of these is portable low fixed-field MRI (typically 60 – 200mT).\(^3\) Studies assessing the impact of this imaging method on stroke diagnosis and treatment are ongoing.\(^4\) The ability to safely measure T\(_1\) relaxation time-constant changes due to cellular level alterations at very low magnetic fields could prove a useful and safe adjunct in assessment and follow up of stroke patients, particularly in rural or low resource settings.\(^5\)

Field-Cycling Imaging (FCI) MRI is a new whole-body imaging technology constructed at the University of Aberdeen, merging MRI and Fast Field-Cycling NMR (FFC-NMR) to explore the magnetic spectrum and measure molecular dynamics in vivo.\(^6\) It undertakes the latter by measuring changes in T\(_1\) relaxation time-constant of tissues over a wide range of magnetic field strengths, rapidly switching between different fields during the pulse sequence. The profile of T\(_1\) as a function of the field B\(_0\), referred to as the T\(_1\) Nuclear Magnetic Relaxation Dispersion (NMRD) profile, closely relates to water dynamics over time scales that complement diffusion-weighted (DWI) MRI.\(^7\)\(^-\)\(^12\) This uniquely captures relaxation mechanisms, reflecting cellular molecular dynamics as illustrated *in vitro* in cartilage,\(^13\)\(^-\)\(^15\) blood,\(^16\) sarcoma,\(^17\) and in animal models of breast\(^18\) and brain malignancy.\(^19\) Whole-body FCI has not previously been reported in the literature.

This proof-of-concept study was therefore developed to test the hypothesis that FCI can differentiate between endogenous T\(_1\) dispersion in sub-acute ischemic stroke and non-ischemic brain at a range of magnetic fields between 0.2mT and 200mT. The aim was to test whether a prototype whole-body FCI scanner can identify infarct regions in patients with subacute ischemic stroke.

**Methods**

This prospective study was approved by the North of Scotland Research Ethics committee (16/NS/0136). Written informed consent was obtained from all participants.
Study participants

Consecutive patients admitted to the acute stroke unit at a University Teaching Hospital between February 2018 to March 2020 and April 2021 to December 2021 were screened. To be eligible for inclusion patients had a documented ischemic stroke on CT or MRI, be able to undergo FCI within seven days of onset, be able to weight bear and have a BMI (Body Mass Index) <28 to be able to access the prototype scanner. Patients were excluded if they had a previous stroke or were unable to give informed consent. Two participants were included in a prior study\textsuperscript{20}. See supplementary material for details.

Imaging Protocols

Participants were scanned using the homebuilt whole-body FCI scanner with a 2-channel, 8-legs quadrature birdcage radiofrequency head coil tuned at 8.2MHz (193mT for protons) for both signal transmission and reception (Figure 1a). As no previous data were available, modifications to optimize the procedure were made as the study progressed resulting in three different configurations (Table 1). FCI images were acquired using a field-cycled inversion recovery spin-echo sequence (Figure 1b) with four to six evolution fields typically ranging logarithmically from 0.2T to 0.2mT, five evolution times (ranging from 5 – 546ms), 20 kHz bandwidth, 8.2 MHz acquisition frequency, in-plane resolution of 2 to 4mm, slice thickness of 10mm, echo time of 16 to 24ms, single-slice acquisition and no signal averaging. Images had a typical field of view of 290 to 320mm and a matrix size of 64 x 64 to 128 x 128. Image acquisitions were accelerated using Partial Fourier, with typical under-sampling ratios of 25 - 40%. Pre-polarisation was done at 200mT for 300ms before each evolution period. The procedure took 45 minutes including calibration scans, acquisition of axial and sagittal navigator images and FCI scans.

Baseline CT images were obtained on an Optimas 660 (GE) and MRI scans on a Philips 3T MRI (dStream). Standard MRI protocols included T\textsubscript{1}, T\textsubscript{2}, fluid-attenuated inversion recovery, diffusion-weighted imaging (DWI) and gradient echo T\textsubscript{2}\* sequences. The infarct region was outlined by an experienced clinician (MJM), and the FCI slice co-registered with the identified region.
Processing and Analysis of FCI images

Raw images were pre-processed to correct for phase encode and ghosting artefacts using in-house software\(^{21}\). \(T_1\) maps were obtained using a joint total generalized variation (TGV)\(^{22}\) developed in Python 3 (version 3.6; Python Software Foundation). \(T_1\) maps obtained below 20mT were used to estimate signal in regions of interest (ROIs) selected manually within the infarct region and a matched region of tissue from the contralateral unaffected hemisphere. \(T_1\) NMRD profiles were extracted from the ROIs for each participant using in-house software written in MATLAB (v2021a, Mathworks) ([FFC-MRI/T1extractor_from_h5file (github.com)](https://github.com)) and fitted using a power law model\(^{23}\):

\[
T_1(B_0^E) = \alpha B_0^E \beta
\]

Equation 1

where \(B_0^E\), \(\alpha\) and \(\beta\) are the evolution field, amplitude and dispersion of the power law model, respectively. In a log-log plot, \(\alpha\) corresponds to the value of the NMRD profile at 1mT and \(\beta\) to the slope of the curve. \(T_1\) dispersion slope maps were obtained using voxel-by-voxel fitting.

The infarct-to-contralateral tissue contrast ratio in \(T_1\) maps was calculated as the percentage difference in \(T_1\) between two regions as:

\[
\%\Delta T_1 = \frac{T_{1,stroke} - T_{1,normal}}{(T_{1,stroke} + T_{1,normal})/2} \times 100
\]

Equation 2

where \(T_{1,stroke}\) and \(T_{1,normal}\) are the \(T_1\) value of the infarct and contralateral brain tissues, respectively.

Signal-to-noise ratio (SNR) and contrast-to-noise ratio (CNR) were measured from FCI images at the longest evolution time for each field (see equations in Supplementary Material).

Two trained independent readers (GG and EF, 10 and 4 years’ experience respectively) were presented with FCI images denoised using the BM3D algorithm\(^{24}\). Readers were blinded to clinical information and asked to record presence and site of a lesion.
Statistical analysis

Statistical analysis was performed by two authors (VM and LB) using SPSS (version 29.0; IBM). A P value of <.05 was used as the threshold of statistical significance. Categorical data are presented as number with percentage; continuous data are presented as mean ± SD for normally distributed data or median with interquartile range for nonnormally distributed data. The slope and scaling factor $\alpha$ of the $T_1$ dispersion profiles were compared using Wilcoxon signed-rank tests, and Spearman’s rank correlation was used to explore the association between infarct-to-contralateral tissue ratio and field strengths, for participants where the infarct was conspicuous. Interrater reliability was calculated using Cohen’s kappa.$^{25}$

As this was an exploratory study, formal power calculation was not performed.

Results

Participant Characteristics

Error! Reference source not found. 685 patients were screened for eligibility. After exclusion of 319 as unable to weight bear, 158 unable or unwilling to consent, 172 with high BMI, 26 were enrolled in the study (Figure 2). Twelve participants could not complete the FCI scan due to technical issues (five) or claustrophobia (seven). Fourteen completed scanning and were analysed for inter-rater agreement. Five of these were excluded from FCI analysis as a single slice missed the level of a small infarct. Thus, data from 9 participants (mean age [SD], 62.3 years ± 15.5; 9 males) were processed for $T_1$ dispersion (Table 2).

Quality of FCI

The typical magnitude images achieved an SNR (CNR) of 12 ± 4 (3.2 ± 1.4) at 0.2T and 2.3 ± 1.0 (1.5 ± 1.1) <1.3mT. There was higher tissue contrast at longer evolution times: for example, Figure 3 (participant 6) shows higher tissue contrast at 136ms than 10.9ms for 2.2mT (CNR: 4.0 and 1.5 respectively). While the infarct was less visible at 200mT, in all participants images ≤37mT exhibited hyper-intense $T_1$ regions corresponding to the region...
identified on baseline imaging. This was less conspicuous in two participants because of low SNR (participants 7 and 8) (Figure 4).

Quality of T1 maps generated from FCI

On T1 maps, infarct-to-contralateral tissue contrast ratio was lowest at 0.2 T and increased with decreasing field strengths (r(26) = -0.68, P < .001, Figure 5a). On example T1 maps from participant 6 (Figure 6), infarct regions were clearly visible at 21.1 and 2.3mT, where the infarct-to-contralateral tissue contrast ratio was largest (12.3% at 0.2T, 46.6% at 21.1mT, 46.2% at 2.3mT).

T1 dispersion profiles

The power law model correctly fitted the T1 NMRD profiles in both infarcted and unaffected tissue (SSE: 0.0019 and 0.0010 respectively). The T1 relaxation time-constant between 0.2T and 0.2mT was higher in infarct regions than the matched region (mean range 612 – 95 ms vs 470 – 36 ms; P range 0.018 – 0.109) (Figure 4b, Figure S1), with an increasing difference as the field decreased (27% at 200mT, 58% at 21.1mT and 182% at 0.2mT). This was reflected in the T1 dispersion, which was smaller in infarct than unaffected tissue (median [IQR]: 0.23 [0.18 – 0.37] vs 0.35 [0.27 – 0.43]; P = .03).

There was no evidence of a difference in the scaling factor α of the T1 NMRD profiles between stroke and contralateral ROIs (median [IQR]: 0.79 [0.53 – 1.2] vs 0.82 [0.50 – 1.0]; P = .13).

Inter-reader Agreement

The percentage of agreement for site of lesion was 85.7% and Cohen’s kappa was 0.69 suggesting substantial agreement^25 (Table S1).

Discussion

The inequity in access to detailed stroke imaging^5 highlights a potential role for dedicated ultra-low field (< 0.2T) scanners which can rapidly and safely detect ischemic stroke. Siting these in ambulances or rural settings could help direct initial or follow-up care^3.
Using a prototype whole-body FCI scanner which can vary magnetic field, we aimed to identify subacute ischemic stroke at field strengths between 0.2mT and 0.2T. We found that the infarct was identifiable in nine participants with substantial inter-reader agreement (Cohen’s Kappa 0.69). $T_1$ relaxation time constant was higher in infarct regions than in contralateral unaffected tissue, but the dispersion was lower ($P = .03$). $T_1$ dispersion maps are shown in Figure S2. The best infarct-to-contralateral tissue contrast ratio for identifying subacute ischemic stroke appears to be below 20mT. Further exploration at fields $<0.2$mT may provide data useful to future design of low-field MRI systems. The shape of the $T_1$ NMRD profile exhibited power law behavior across participants and tissues (SSE of 0.0019 in infarct and 0.001 in unaffected brain). Interestingly, FCI can differentiate the average water content of tissue, which appears as a general scaling factor in the dispersion profile, from a change in water containment which modifies the dispersion.

Based on our proof-of-concept findings, we suggest that identifying field strengths and evolution times which optimize $T_1$ contrast in brain might allow design of low-field devices with a tailored pulse sequence at a single optimal field strength (e.g. between 2 and 0.2mT). These would be able to acquire diagnostic images within 2-3 minutes. To further characterize tissues by their water dynamics using NMRD and $T_1$ dispersion slope maps might require identification of 3 to 4 optimal fields and approximately 5 evolution times: these would take about 10 minutes to acquire. On a dedicated scanner, this could considerably speed up the acquisition. We also anticipate a diagnostically useful role for in vivo $T_1$ contrast measurement in many other diseases such as cancer, bone, and lung disease.

There were several limitations. First, in this proof-of-concept study on a prototype scanner, we were only able to include a small number of participants, some of whom had small infarcts. Secondly, the reference tissue ROI may also have been composed of both white and grey matter due to the low resolution of FCI, increasing variation in $T_1$. Thirdly, we observed significant inter-participant variability in $\%\Delta T_1$ with increasing field strength. This may reflect a combination of technical factors: inconsistent SNR performance arising from the FCI scanner prototype, protocol changes reducing statistical power, and/or motion artefact, and participant factors: infarct volume or differences in ischemic tissue structure related to time since ictus, oedema and degree of cell death. A better-defined tissue reference taking 3T MRI
images and time from ictus into consideration may provide lower inter-participant variability in the contrast profiles and allow more precise estimates of $T_1$ dispersion.

In conclusion, this study demonstrates that FCI can identify sub-acute ischemia at field strengths down to 0.2mT. Since completing this study, we have developed an improved FCI prototype to confirm our results, adding participants with intracerebral hemorrhage and controls. $T_1$ NMRD may provide additional information to low fixed-field MRI and this warrants further study in other neurological conditions and diseases.
References


**Table 1: Field Cycling Imaging acquisition details.**

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<th>Protocol ID</th>
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<tr>
<td>37</td>
<td>196</td>
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<td>63 36 21 12</td>
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<td>36</td>
<td>27 16 9 5</td>
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<td>2</td>
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<tr>
<td>21.1</td>
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<td>169 86 44 22</td>
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<td>158</td>
<td>80 41 21 11</td>
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<tr>
<td>0.2</td>
<td>75</td>
<td>38 19 10 5</td>
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Notes: Over the course of the study, three modifications were made to the evolution fields and times.
Table 2: Participant Characteristics.

<table>
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<tr>
<th>ID</th>
<th>Age (y)</th>
<th>Stroke location</th>
<th>Side</th>
<th>NIHSS score</th>
<th>Modified Rankin Score</th>
<th>Scan time after onset (days)</th>
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<td>2</td>
<td>2</td>
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<tr>
<td>3</td>
<td>67</td>
<td>Occipital lobe</td>
<td>right</td>
<td>1</td>
<td>2</td>
<td>5</td>
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<td>3</td>
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<td>5</td>
<td>55</td>
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<td>1</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
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<td>Occipital lobe</td>
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<td>3</td>
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<td>Cingulum</td>
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<td>2</td>
<td>4</td>
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<tr>
<td>8</td>
<td>84</td>
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<td>9</td>
<td>57</td>
<td>Insula</td>
<td>right</td>
<td>1</td>
<td>2</td>
<td>1</td>
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</table>

Mean ± SD  62.3 ± 15.5

Notes — NIHSS (National Institutes of Health Stroke Scale) and Modified Rankin Scores are given from the time of recruitment. All included participants were male.
Figure 1: a) Photograph of the FCI scanner prototype, with the head coil in place. b) Timing diagram of the FCI inversion recovery sequence used for this study. The magnetic field is switched rapidly between different field levels, polarization ($B_0^P$), evolution ($B_0^E$) and detection ($B_0^D$) to prepare the contrast levels for image acquisition. Polarization is done at higher field to increase the image signal, evolution at low field and detection at higher field. After polarization an inversion pulse is applied before relaxation occurs at pre-selected evolution fields $B_0^E$ for an evolution time (see arrow). Signal detection is performed using a conventional spin echo acquisition (gradient lines GR, GP and GS). The whole process is repeated for different evolution times and fields to estimate the $T_1$ Nuclear Magnetic Resonance Dispersion profiles. Note that FCI images are always acquired at the same detection field, allowing a single radiofrequency coil to be used for the entire scan.

Figure 2. Flow chart of study participants.

Figure 2: Axial Field Cycling Imaging (FCI) in a 79-year-old male participant with right occipital lobe infarct (participant 6) with no contrast agent used. Images were acquired at three magnetic field strengths (rows) with five evolutions times $t_E$ (columns), 6 days after onset. The infarct is most visible at lower acquisition field strengths (at 21.1mT and 2.2mT) and longer evolution times (arrows). The small dot below the skull is a positioning marker. The infarct is detected using endogenous $T_1$ contrast mechanisms.

Figure 3: Comparison of Field Cycling Imaging (FCI) with conventional imaging. 3T MRI, FCI and $T_1$ maps are shown for all participants. The infarct region in FCI images and $T_1$ maps (shown in yellow) corresponded with the infarct observed on baseline diagnostic scans (MRI/CT). Participant 6 did not have a 3T MRI performed. The infarct was less conspicuous in participants 7 and 8 due to low SNR. The color bars in $T_1$ maps show the $T_1$ values across the brain from low (blue) to high (yellow) values and are expressed in ms.

Figure 4: (A) infarct to matched contralateral tissue contrast ratio ($\%\Delta T_1$) obtained from the $T_1$ maps versus evolution field for each participant ($n = 9$), and (B) Line plots show $T_1$ Nuclear Magnetic Resonance Dispersion (NMRD) profiles. The $\%\Delta T_1$ and the profiles were processed from regions of interest (ROI) taken in the infarct area and matched brain tissue from the
unaffected hemisphere. The $\%\Delta T_1$ is plotted as a function of the evolution field where the evolution field is in a log scale. The $T_1$ NMRD profiles are shown in a log-log plot. The infarct to matched contralateral tissue contrast ratio increased with decreasing field strengths between 0.2T and 0.2mT. Infarct areas demonstrated higher $T_1$ relaxation time constant than the unaffected brain areas with their differences increasing at lower field strengths.

Figure 6: $T_1$ maps generated after processing the FCI images in a 79-year-old male participant with right occipital lobe infarct (participant 6). Images were processed with a TGV regularization. The infarct, shown in yellow, demonstrates higher $T_1$ relaxation time constant than the unaffected brain tissue. The infarct is clearly visible at 21.1 and 2.2mT where the infarct-to-contralateral tissue contrast ratio is higher ($\%\Delta T_1$ of 12.3% at 0.2 T, 46.6% at 21.1mT, 46.2% at 2.3mT). The color bars are the $T_1$ values in ms.
Supplemental Material

The signal-to-noise ratio (SNR) was measured from the FCI images at the longest evolution time for each field using the equation:

\[ \text{SNR} = 0.66 \times \frac{S_{\text{normal}}}{\text{Noise}} \]

where \( S_{\text{normal}} \) is the mean intensity from the ROI in the contralateral area and Noise is the standard deviation from the ROI on background noise. The 0.66 correction factor is included to account for the non-Gaussian noise distribution.

The contrast-to-noise ratio (CNR) was measured from the FCI images at the longest evolution time for each field as:

\[ \text{CNR} = 0.66 \times \frac{S_{\text{stroke}} - S_{\text{normal}}}{\text{Noise}} \]

where \( S_{\text{stroke}} \) is the mean signal intensity from the ROIs in infarct area.

Figure S1: Line plots of \( T_1 \) Nuclear Magnetic Resonance Dispersion profiles from each of the nine participants with ischemic stroke. The \( T_1 \) values were calculated for the region of interest (ROI) in the infarct areas (red circles) and contralateral matched unaffected brain tissue (blue squares) for each participant. The error bars show the standard deviation within the ROIs. The lines show the linear fitting in a log-log scale.

Figure S2. \( T_1 \) dispersion slope maps from participants with ischemic stroke (participants 7 and 8 excluded due to small lesions). \( T_1 \) dispersion values were calculated by voxel-by-voxel fitting of the \( T_1 \) Nuclear Magnetic Resonance Dispersion profiles using a power law model. The black arrow shows the infarct area (blue in color). Maps are noisy and the infarct area is not always clearly visible. The color bar shows the slope values.

Participant Overlap

2 (participants 4 and 6) were included in a methodological paper describing \( T_1 \) dispersion image reconstruction using total generalized variation regularization. This prior article dealt
with development of the methodology of image analysis and interpretation whereas in this
manuscript imaging quality and ability to visualize infarct regions is assessed.
Table S1: Interreader agreement for presence of stroke lesion

<table>
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<tr>
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<th>MRI or CT lesion on matched FCI slice</th>
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<tbody>
<tr>
<td></td>
<td>Lesion present</td>
<td>No lesion</td>
</tr>
<tr>
<td>FCI reader B</td>
<td>Lesion present</td>
<td>9*</td>
</tr>
<tr>
<td></td>
<td>No lesion</td>
<td>0</td>
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Notes.-- 9 scans with lesion (2 had poor Signal Noise Ratio), 5 scans with no stroke lesion.

*The percentage of agreement for whether a lesion was present was 92.86% (Cohen’s kappa 0.84) suggesting almost perfect agreement\(^2\). However, for one case with poor SNR where both raters agreed a lesion was present, there was disagreement over the site of the lesion. When this was included in analysis the percentage agreement was 85.7% and Cohen’s kappa was 0.69 suggesting substantial agreement.
Author contributions

LB contributed to the funding and design of the study, patient scans, study coordination, data analysis and to the writing of the manuscript; JR contributed to the design of the study, patient scans, data analysis and to the writing of the manuscript; VM contributed to the patient scans, data analysis and to the writing of the manuscript; OM conducted the TGV analysis; GGG contributed to patient recruitment and data interpretation; EF contributed to the image interpretation; DL contributed to the funding and design of the study, and to the writing of the manuscript; MJM contributed to the funding and design of the study, ethics submission, patient recruitment, study coordination, image interpretation and to the writing of the manuscript.

Acknowledgements

The authors would like to thank radiographers Nichola Crouch, Mike Hendry, Laura Reid, Michelle Mauchline, and Arthur Ginsburg for their support with patient scans of FCI, Stacey Dawson for the study coordination, and the Scottish Stroke Research Network nurses, Janice Irvine, Annika Walch, Farah Muir and Sandra Williams who helped with patient recruitment. We also thank Alison Murray who contributed to study set up and 3T image interpretation. Funding for the scanner prototype was obtained from the European Union’s Horizon 2020 research and innovation programme under grant agreement No 668119 (project “IDentIFY”) and funding for VM during write up came from the Chief Scientist Office (Grant TCS/19/44).
Figure 1: a) Photograph of the FCI scanner prototype, with the head coil in place.

90x70mm (300 x 300 DPI)
Figure 1b.tif Timing diagram of the FCI inversion recovery sequence used for this study. The magnetic field is switched rapidly between different field levels, polarization ($B_0^P$), evolution ($B_0^E$) and detection ($B_0^D$) to prepare the contrast levels for image acquisition. Polarization is done at high field to increase the image signal, evolution at low field and detection at high field. After polarization an inversion pulse is applied before relaxation occurs at pre-selected evolution fields $B_0^E$ for an evolution time (see arrow). Signal detection is performed using a conventional spin echo acquisition (gradient lines GR, GP and GS). The whole process is repeated for different evolution times and fields to estimate the $T_1$ NMRD profiles. Note that FCI images are always acquired at the same detection field, allowing a single radiofrequency coil to be used for the entire scan.

265x190mm (600 x 600 DPI)
Figure 2. Flow chart of study participants.

156x110mm (300 x 300 DPI)
Figure 3: Axial Field Cycling Imaging (FCI) in a 79-year-old male participant with right occipital lobe infarct (participant 6) with no contrast agent used. Images were acquired at three magnetic field strengths (rows) with five evolutions times $t_e$ (columns), 6 days after onset. The infarct is most visible at lower acquisition field strengths (at 21.1mT and 2.2mT) and longer evolution times (arrows). The small dot below the skull is a positioning marker. The infarct is detected using endogenous $T_1$ contrast mechanisms.
Figure 4: Comparison of Field Cycling Imaging (FCI) with conventional imaging. 3T MRI, FCI and T1 maps are shown for all participants. The infarct region in FCI images and T1 maps (shown in yellow) corresponded with the infarct observed on baseline diagnostic scans (MRI/CT). Participant 6 did not have a 3T MRI performed. The infarct was less conspicuous in participants 7 and 8 due to low SNR. The color bars in T1 maps show the T1 values across the brain from low (blue) to high (yellow) values and are expressed in ms.

170x368mm (300 x 300 DPI)
Figure 5a: Infarct to matched contralateral tissue contrast ratio (%ΔT₁) obtained from the T₁ maps versus evolution field for each participant (n = 9).

148x111mm (300 x 300 DPI)
Figure 5b: Line plots show $T_1$ Nuclear Magnetic Resonance Dispersion (NMRD) profiles. The $\% \Delta T_1$ and the profiles were processed from regions of interest (ROI) taken in the infarct area and matched brain tissue from the unaffected hemisphere. The $\% \Delta T_1$ is plotted as a function of the evolution field where the evolution field is in a log scale. The $T_1$ NMRD profiles are shown in a log-log plot. The infarct to matched contralateral tissue contrast ratio increased with decreasing field strengths between 0.2T and 0.2mT. Infarct areas demonstrated higher $T_1$ relaxation time constant than the unaffected brain areas with their differences increasing at lower field strengths.

143x113mm (300 x 300 DPI)
Figure 6: T₁ maps generated after processing the FCI images in a 79-year-old male participant with right occipital lobe infarct (participant 6). Images were processed with a TGV regularization. The infarct, shown in yellow, demonstrates higher T₁ relaxation time constant than the unaffected brain tissue. The infarct is clearly visible at 21.1 and 2.2 mT where the infarct-to-contralateral tissue contrast ratio is higher (%ΔT₁ of 12.3% at 0.2 T, 46.6% at 21.1 mT, 46.2% at 2.3 mT). The color bars are the T₁ values in ms.
Figure S1: Line plots of T1 Nuclear Magnetic Resonance Dispersion profiles from each of the nine participants with ischemic stroke. The T1 values were calculated for the region of interest (ROI) in the infarct areas (red circles) and contralateral matched unaffected brain tissue (blue squares) for each participant. The error bars show the standard deviation within the ROIs. The lines show the linear fitting in a log-log scale.
Figure S2. T1 dispersion slope maps from participants with ischemic stroke (participants 7 and 8 excluded due to small lesions). T1 dispersion values were calculated by voxel-by-voxel fitting of the T1 Nuclear Magnetic Resonance Dispersion profiles using a power law model. The black arrow shows the infarct area (blue in color). Maps are noisy and the infarct area is not always clearly visible. The color bar shows the slope values.

289x243mm (300 x 300 DPI)
Field-Cycling Magnetic Resonance Imaging for identifying Minor Ischemic Stroke below 0.2 tesla.

- In this prospective study of 9 participants with sub-acute ischemic stroke, infarct regions were identified using endogenous T1 contrast mechanisms at field strengths between 200mT and 0.2mT with inter-rater agreement of 85.7%, Cohen’s kappa 0.69.

- T1 relaxation time-constants at ultra-low fields between 0.2T and 0.2mT were higher in infarct regions than in contralateral unaffected brain tissue (mean range 612 – 95ms vs 470 – 36ms; P range .018 – .109).