Radiology

Activity for 2020

Activity No: A13 (20) 2023

Topic
Intracranial vessel wall imaging

Article
The use and pitfalls of intracranial vessel wall imaging: how we do it

Speciality
CT / MRI / Ultrasound

Approved for THREE (3) Clinical Continuing Educational Units (CEU’s)
The Use and Pitfalls of Intracranial Vessel Wall Imaging: How We Do It

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Intracranial vessel wall magnetic resonance (MR) imaging has gained much attention in the past decade and has become part of state-of-the-art MR imaging protocols to assist in diagnosing the cause of ischemic stroke. With intracranial vessel wall imaging, vessel wall characteristics have tentatively been described for atherosclerosis, vasculitis, dissections, Moyamoya disease, and aneurysms. With the increasing demand and subsequently increased use of intracranial vessel wall imaging in clinical practice, radiologists should be aware of the choices in imaging parameters and how they affect image quality, the clinical indications, methods of assessment, and limitations in the interpretation of these images. In this How I do It article, the authors will discuss the technical requirements and considerations for vessel wall image acquisition in general, describe their own vessel wall imaging protocol at 3 T and 7 T, show a step-by-step basic assessment of intracranial vessel wall imaging as performed at their institution—including commonly encountered artifacts and pitfalls—and summarize the commonly reported imaging characteristics of various intracranial vessel wall diseases for direct clinical applicability. Finally, future technical and clinical considerations for full implementation of intracranial vessel wall imaging in clinical practice, including the need for histologic validation and acquisition time reduction, will be discussed.

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In recent years, intracranial vessel wall (VW) magnetic resonance (MR) imaging has seen an exponential increase in popularity and clinical applicability (1,2). Several years ago, VW imaging was restricted to extracranial (peripheral) arteries, such as the carotid arteries, in which atherosclerotic “vulnerable plaques”–prone to causing embolism and subsequent ischemic stroke–could be assessed by using different dedicated MR imaging sequences. However, increasing evidence shows that not extracranial but intracranial atherosclerosis is the leading cause of ischemic stroke worldwide (3–6). In addition, intracranial atherosclerosis has been associated with an increased (recurrent) stroke risk and vascular dementia (4,7–9). This change of insight into causes of ischemic stroke has led to a trend toward imaging the intracranial vasculature.

Traditional imaging methods for visualizing the intracranial arteries include intra-arterial digital subtraction angiography (DSA), computed tomographic (CT) angiography, Doppler ultrasonography (US), and MR angiography. DSA can depict stenotic lesions in large, as well as small arteries, including the A2 segment of the anterior cerebral artery, M2-M3 segments of the middle cerebral artery (MCA), P2 segment of the posterior cerebral artery, and even more peripheral segments of the intracranial arteries. With unenhanced CT, arterial VW calcifications can be detected that are associated with future stroke risk (10–13). CT angiography is an easily accessible and fast procedure in patients with acute stroke and can depict stenotic lesions very accurately in the more proximal intracranial arteries and its branches (14). Doppler US can depict the VW of primarily the MCA, thereby providing (single-artery restricted) information on VW disease. Finally contrast material–enhanced and time-of-flight MR angiography, like CT angiography, can depict stenotic lesions in proximal cerebral arteries, less quickly but without additional radiation dose compared with CT angiography. However, all of these techniques, except for Doppler US, have the disadvantage of only depicting the arterial lumen (including possible stenoses) and not the culprit VW itself. Intracranial VW imaging has two major advantages over DSA, CT angiography, and MR angiography: it can depict nonstenotic lesions and it can further characterize stenotic lesions that have already been detected with common angiographic methods.

In this How I do It article, we describe the utility and practical steps for performing and interpreting intracranial VW MR images. These practical steps are based on our teaching experience of intracranial VW imaging to radiologists and neuroradiologists who have started using this method in daily clinical practice. The key questions that are often raised during their learning curve will also be addressed in this article. Key steps and common VW pathologic conditions will be shown in illustrative figures of patient examples.

**The Imaging Protocol–What Do We Need?**

**VW Imaging Sequence Prerequisites**

For intracranial VW imaging, both a high contrast-to-noise ratio (CNR) and a high spatial resolution are needed to visualize the thin arterial VW and to characterize VW lesions. CNR in VW imaging comprises signal contrast of the VW relative to its direct surroundings, that is, blood and cerebrospinal fluid (CSF). It is dependent on both sequence parameters and magnetic field strength, where CNR generally increases with increasing field strength. The achievable spatial resolution is also dependent on the field strength, as well as on the acquisition time. Finally, the different image contrast weightings need to be considered when developing a VW imaging protocol (1).

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**Essentials**

- The main advantages of intracranial vessel wall (VW) imaging over luminal-based methods are detection of nonstenotic lesions and further characterization of stenotic lesions that have already been detected with common angiographic methods.

- For intracranial VW imaging, a minimum magnetic field strength of 3 T is necessary to obtain a sufficient contrast-to-noise ratio (CNR) and spatial resolution to visualize the thin arterial VW and to characterize VW lesions.

- Radiologists should be acquainted with the clinical patient information and the applied imaging parameters for an optimal image assessment.

- Radiologists benefit from a systematic approach in VW assessment.

- Difficulties in assessing VW images are 
  
  (a) variances in wall thickness not related to VW disease; (b) close proximity of the VW to the brain parenchyma or enhancing cavernous venous plexus; (c) nonpathologic contrast-enhancing areas of the VW when crossing the dura mater; and (d) artifacts due to motion, sensitivity encoding fold-over, free induction decay, or slow flow.

- Technical challenges include reducing acquisition time to perform both pre- and postcontrast sequences without substantial decrease in signal-to-noise ratio and CNR and histopathologic validation of VW imaging.
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VW CNR.—The first step in achieving a high VW CNR is to suppress the luminal blood. Most intracranial VW imaging sequences rely on the intrinsic “black blood” properties of three-dimensional (3D) turbo spin-echo (TSE) pulse sequences, with variable flip-angle refocusing pulses (17,19–23). In these sequences, black blood is achieved by intravoxel dephasing of flowing blood, which is most effective when low-flip-angle refocusing pulses are used (17,24). Alternatively, preparation pulses can be used to obtain blood suppression, such as double inversion recovery (25), motion-sensitizing preparation pulses (26), or delay alternating with nutation for tailored excitation (DANTE) preparation pulses (20). Adding preparation pulses generally increases acquisition time. Currently, there is a need for a thorough analysis, both theoretically and experimentally, assessing the performance of the various blood suppression techniques in the presence of slow flow (directly next to the VW).

For optimal CNR, not only suppression of blood signal to depict the inner boundary of the VW, but also suppression of CSF signal to depict the outer boundary is essential, especially when outward vascular remodeling is present (27–30). At 3 T, the CNR between VW and lumen is superior to the CNR between VW and CSF on most pulse sequences. Because an inversion-recovery pulse as a CSF-suppressing technique is both time consuming and detrimental to the VW signal-to-noise ratio (SNR), these VW imaging sequences rely implicitly (without use of CSF suppression preparation pulses) or explicitly (with use of DANTE) on CSF flow for CSF suppression (19–21,31,32). Consequently, suppression of CSF will be lower in compartments where there is a slower flow of CSF, for example, around the VW and at locations where there is little CSF surrounding the vessels. However, a recent promising development for improving CSF suppression at 3 T is the incorporation of an antidriven-equilibrium pulse, which relies on T1 and T2 relaxation properties and is independent of CSF flow (32,33). At higher field strengths, such as 7 T, the higher attainable SNR enables the use of previously mentioned inversion-recovery pulse, resulting in nearly optimal CSF suppression (15).

Spatial resolution.—Due to a lack of in vivo-ex vivo correlation studies (see “Future Prospects”), it is currently not clear what minimum spatial resolution is necessary to accurately diagnose intracranial VW disease, nor is it known what spatial coverage is clinically relevant. Previous histopathologic studies have shown the intracranial arterial VW to vary in thickness from 0.2 to 0.4 mm for the distal internal carotid artery to 0.2–0.3 mm for the MCA (34). However, radiologic measurements have shown a larger VW thickness of approximately 1.0 mm for both MCA and internal carotid artery (35). Several reasons can account for this difference; for example, underestimation in histopathologic studies due to preparation techniques (cell shrinkage of 8%–20% [36,37] in histopathologic preparation) or overestimation in the radiologic studies due to partial volume effects (relatively large voxels), measurement errors, and/or pulsatility effects of the VW during acquisition. On the basis of these considerations, and given the fact that one needs at least two voxels in an object to measure its size and/or thickness accurately (38), an MR imaging sequence with 0.18-mm isotropic voxels—assuming a histologically processed wall thickness of 0.3 mm and a mean shrinkage effect of 15%—would theoretically enable highly detailed assessment of the circle of Willis and, depending on the field of view (FOV), its large and small branches (34). However, it remains to be seen whether this much detail is really necessary in clinical practice in all patient groups (39). Also, (ultra)high-resolution sequences would be met with significant time constraints, limiting their application to cooperative patients with low morbidity or necessitating either introduction of very fast imaging techniques or compromising between spatial resolution and FOV.

To avoid these limitations, intracranial VW imaging sequences in current practice are mainly aimed at detecting larger lesions in the intracranial arteries proximal and just distal to the circle of Willis (M1, A1, P1 segments) and at the border of the M1-M2, A1-A2, and P1-P2 segments. The acquired in-plane spatial resolution of these sequences ranges between 0.4 × 0.4 mm² and 0.9 × 0.9 mm² (1,18,28,31,35,40,41). For more distal arteries, however, detection reliability will decrease because the diameter of these arteries and therefore lesions at the walls of these arteries become smaller, increasing the impact of partial volume effects (35), which needs to be taken into account when assessing the VWs in clinical practice (see “Systematic Approach of VW Assessment”).

Isotropic versus anisotropic voxels.—A related question is whether and/or when to use isotropic or anisotropic voxels. Most experts in the field of VW imaging sequence development prefer sequences with isotropic voxels, which render multiplanar assessment more feasible. Compared with, for example, the carotid artery, intracranial arteries are often tortuous and have varying orientations, making multiplanar reconstruction an important asset in assessment of these arteries. In anisotropic sequences, a very high in-plane spatial resolution can be achieved within reasonable acquisition times, enabling detailed assessment of VW lesions and atherosclerotic plaque characterization when the FOV is placed perpendicular to the lesion. However, due to the larger through-plane voxel size, small lesions are subject to partial volume effects. Therefore, it may well be dependent on the specific clinical question on a single-patient basis which type of sequence (isotropic versus anisotropic voxels) to use. For instance, a radiologist may consider isotropic sequences in patients with no previous imaging or known VW lesion as a method of screening the intracranial arteries, while an anisotropic sequence could be used to assess a known lesion.

The advantage of interpreting VW images in multiple planes is currently not supported by much research data. The results of recent studies suggest that the use of transverse images alone may be
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sufficient for the interpretation of VW images (42,43). In one of these studies, 3D sequences with a high in-plane spatial resolution and a larger section thickness were either performed in the transverse plane or perpendicular to the MCA. VW lesions of the MCA were also detected on the images that were acquired nonperpendicular to the artery.

**Two-dimensional versus 3D imaging.**—To increase in-plane spatial resolution, two-dimensional (2D) spin-echo sequences can be used as an alternative to 3D methods (1,28,31,44). The 2D in-plane spatial resolution is equivalent to that of 3D, and volume-averaging errors due to thicker sections (2 mm, no gap) are alleviated by repeating the sequences in sagittal and/or axial planes targeted to a lesion in question. Sequence acquisition times are short (approximately 3 minutes) with good SNR. The drawbacks of these 2D spin-echo methods are the need to target a specific lesion due to limited coverage to keep imaging times short and the increased dependence on correct positioning of the FOV. Two-dimensional methods can therefore best be applied in cases in which there is a known intracranial stenosis on which the FOV can be focused and has highest value in characterizing the underlying pathologic process (eg, atherosclerotic plaque characterization).

**Field strength.**—A further consideration is what field strength to use. In our experience and that of others (2), 1.5 T does not achieve sufficient SNR and CNR within a reasonable acquisition time. Therefore, a field strength of 3 T or higher is mandatory. In light of the increased availability of 3-T MR imaging platforms, this makes intracranial VW imaging feasible in many hospitals worldwide. The advantage of a 7-T field strength is an increased SNR and CNR compared with 3 T, which enables better intrinsic image contrast and a higher spatial resolution within a reasonable acquisition time (16,18). Specifically for VW imaging, the higher SNR also enables the use of the inversion-recovery pulse mentioned above to completely suppress the CSF and a large FOV for whole-brain coverage; however, this does come at the cost of a lower spatial resolution to keep acquisition times within limits. Nonetheless, although current 7-T VW imaging pulse sequences have a lower or comparable spatial resolution compared with published (and our own) 3-T sequences, visualization of the VW was still found superior at 7 T, mainly due to higher CNR and more optimal CSF suppression (45). However, only approximately 60 7-T MR imaging platforms exist worldwide. Also, the inhomogeneous B₀ and B₁ fields can still hamper assessment of peripheral parts of the brain, such as in the Sylvian fissure, although this is improving over time due to the continuous development of the technology. Combined with the fact that 7-T MR imaging has not yet been officially approved for clinical diagnostic imaging, 7-T VW imaging sequences are mainly used for research purposes and specific challenging clinical cases (eg, cerebral vasculitis).

**Image contrast weighting.**—Mirroring pulse sequence development in the extracranial carotid artery, pulse sequences with different image contrast weighting (eg, T₁-, T₂-, and proton density weighted) have also been developed for assessing the intracranial VW. Theoretically, the ideal VW imaging protocol would include images of all three image contrast weightings and a T₁-weighted sequence after contrast agent administration to assess intracranial VW disease in the same fashion as has been done for years for their extracranial counterparts (46). However, no clear evidence yet exists on the clinical relevance of multicontrast MR imaging protocols for intracranial VW disease. With the relatively long acquisition times of these VW sequences, most of the currently used intracranial VW imaging protocols rely on T₁-weighted imaging, because of its benefits in the detectability of contrast enhancement after contrast agent administration and favorable imaging characteristics when distinguishing the VW from its surrounding tissue and (if present) plaque components (47).

**Our 3-T Protocol**

The 3-T MR imaging systems at our center (Achieva 3.0T; Philips Healthcare, Best, the Netherlands) were recently equipped with a 32-channel phased-array sensitivity encoding head coil. Previously, we used an eight-channel head coil. We clinically use a 3D T₁-weighted volumetric isotropically reconstructed TSE acquisition, or VIRTA, sequence (adapted from Qiao et al) (17,42) that is performed after contrast material administration only (gadobutrol, Gadovist 1.0 mmol/mL, single dose, adjusted to patient weight; Bayer Schering Pharma, Newbury, England). The images are acquired at an anisotropic spatial resolution of 0.5 × 0.5 × 1.0 mm³ that is subsequently reconstructed to 0.5 × 0.5 × 0.5 mm³ isotropic resolution. We have chosen this particular spatial resolution, which is considerably below the “ideal” spatial resolution of 0.18 × 0.18 × 0.18 mm³ mentioned above, because of the limitations regarding acquisition time and SNR. Also, in a recent small study, we found that sequences with a lower spatial resolution (0.5 mm × 0.5 mm × 1.0 mm) and a short imaging duration (4 minutes 39 seconds) have a good subjective quality score and good performance with respect to lesion detection (39). An anisotropic sequence was chosen for two main reasons: (a) In our experience, assessment of the in-plane sections, planned in a transverse oblique plane to image the circle of Willis in the classic anatomic way (Fig 1), most often will suffice for detection of larger lesions and (b) we mainly use this sequence to detect VW enhancement, which is less dependent on partial volume effects. In this regard, an anisotropic voxel size can in our opinion be a good compromise with an increase in SNR and shorter acquisition times. However, the above-mentioned drawbacks of anisotropic sequences always need to be taken into account when assessing the images.

In the VIRTA sequence, black blood is obtained by means of low-flip-angle refocusing pulses. To optimize CSF suppression, we use the antidriven-equilibrium method (17,33,39). Because CSF suppression is not optimal with use of this method, we have currently started implementing DANTE for better CSF suppression. Because the FOV of the sequence is
restricted in the through-plane direction—
measuring 45 mm thickness—care must
be taken to plan the FOV correctly (Fig
1). Key parameters of our 3-T protocol
are shown in Table 1.

Our 7-T Protocol

The 7-T MR imaging platform at our
institution (Philips Healthcare, Cleve-
land, Ohio) is equipped with a 32-chan-
el receive coil and a volume transmit/
receive coil for transmission (Nova
Medical, Wilmington, Mass). We use
a T1-weighted magnetization-prepared
inversion-recovery TSE intracranial
VW sequence (15,48) performed be-
fore and after contrast material ad-
ministration, with acquired isotropic
resolution of 0.8 × 0.8 × 0.8 mm³
that is reconstructed to 0.49 × 0.49
× 0.49 mm³ isotropic resolution. The
pulse sequence includes an inversion
recovery pulse that results in nearly
complete CSF suppression and uses
low and varying refocusing pulse angles
for obtaining black blood. The FOV en-
ccompasses the entire brain (190 mm),
which, in combination with the isotro-
ic spatial resolution, does not require
additional planning in oblique angles. B₀
shimming and dielectric pads are addi-
tionally used to decrease inhomogene-
ity of the main magnetic and radiofre-
cquency transmit field, which otherwise
can result in considerable signal loss in
both temporal lobes (including the M2-
M3 trajectory) (49). Key parameters of
our 7-T protocol are shown in Table 2.

Use of Contrast Agents

The use of contrast material–enhanced
MR imaging is considered a necessary
component of VW imaging protocols
(1,2). Sensitivity for contrast enhance-
tment depends on optimal timing of
contrast agent injection. Currently, no
head-to-head comparisons between VW
imaging sequences at different time
points after contrast agent injection
have been performed. For timing of con-
trast enhancement at standard brain im-
ageing sequences, results from studies
in brain tumors are generally used (50). As
a general rule, optimal timing of sequence
acquisition is between approximately
5–10 minutes after contrast agent injec-
tion; contrast enhancement may be weak
within the first 5 minutes after injection,
and evidence of the added value of a late
phase acquisition (> 10 minutes) is lack-
ing. Because many of the current VW
imaging sequences have an acquisition
duration of between 5 and 7 minutes, the
ideal start time would be 5 minutes after
contrast agent injection. At our institu-
tion, we either perform one anatomic
sequence after contrast agent injection—
such as an axial T2-weighted TSE or
diffusion-weighted imaging sequence—or
implement a 5-minute break before start-
ing the VW imaging sequence.

The choice of whether or not to ob-
tain both pre- and postcontrast images
depends on both the specific setting and
clinical question. At 7 T, we perform
the VW imaging sequence before and
after contrast material administration,
because we mainly examine patients in
a research setting or challenging cases
and therefore have time (45–60-minute
time slot) to acquire both pre- and
postcontrast images in addition to the
standard anatomic brain images. With
this setup, contrast-enhancing lesions
can be distinguished from nonenhanc-
ing lesions with a high signal intensity
by comparing pre- and postcontrast
images. However, within the context
of fixed, shorter MR imaging time slots
and a maximum acquisition time based
on patient comfort, in clinical practice,
it is often necessary to limit the time
for intracranial VW imaging at 3 T.
An option that reduces total protocol
time, and which we currently use at our

A Sagittal view of the anterior circulation intracranial arteries and, B, corresponding phase-con-
trast angiographic image used in our clinic for planning the FOV (45-mm feet-head, red box) in a transverse
oblique plane to include all large intracranial arteries of the anterior circulation. C, Transverse view of the
large intracranial arteries of the circle of Willis and, D, corresponding transverse oblique 3-T T1-weighted
intracranial VW MR image, in which most anterior circulation arteries can be seen in one section. A1 and A2
= segments of the anterior cerebral artery; ACOM = anterior communicating artery; BA = basilar artery; ICA
= internal carotid artery; M1, M2, and M3 = segments of the MCA; P1 and P2 = segments of the posterior
cerebral artery; PCOM = posterior communicating artery; VA = vertebral artery.

Figure 1:  

B

C

D

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Table 1

3-T VW Imaging Protocol Used at Our Institution

<table>
<thead>
<tr>
<th>Imaging Parameter</th>
<th>Diffusion-weighted Imaging</th>
<th>3D TOF MR Angiography</th>
<th>2D T2 FLAIR</th>
<th>3D T1 VRITA with Contrast Agent</th>
<th>3D T1 TFE with Contrast Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>FOV (mm²)</td>
<td>230 × 230 × 140</td>
<td>200 × 200 × 70</td>
<td>230 × 183 × 140</td>
<td>200 × 167 × 45</td>
<td>230 × 230 × 160</td>
</tr>
<tr>
<td>Acquisition orientation</td>
<td>Transverse</td>
<td>Transverse</td>
<td>Transverse</td>
<td>Transverse oblique</td>
<td>Transverse</td>
</tr>
<tr>
<td>Acquisition spatial resolution (mm²)</td>
<td>0.96 × 1.22 × 4.0</td>
<td>0.4 × 0.7 × 1.0</td>
<td>0.65 × 0.85 × 4.0</td>
<td>0.5 × 0.5 × 1.0</td>
<td>1.0 × 1.0 × 1.0</td>
</tr>
<tr>
<td>Reconstructed spatial resolution (mm³)</td>
<td>0.45 × 0.45 × 4.0</td>
<td>0.36 × 0.36 × 0.5</td>
<td>0.41 × 0.41 × 4.0</td>
<td>0.5 × 0.5 × 0.5</td>
<td>0.96 × 0.96 × 1.0</td>
</tr>
<tr>
<td>TR/TE/TI (msec)</td>
<td>4056/68/-</td>
<td>22/3.5/-</td>
<td>10000/120/2800</td>
<td>1500/32/-</td>
<td>8.5/3.9/1016</td>
</tr>
<tr>
<td>Flip angle (degrees)</td>
<td>90</td>
<td>16</td>
<td>120</td>
<td>90</td>
<td>8</td>
</tr>
<tr>
<td>Echo spacing (msec)</td>
<td>...</td>
<td>...</td>
<td>9.6</td>
<td>4.5</td>
<td>...</td>
</tr>
<tr>
<td>MPTR TSE factor</td>
<td>...</td>
<td>...</td>
<td>24</td>
<td>56 + 4</td>
<td>...</td>
</tr>
<tr>
<td>Oversampling factor</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>1.8</td>
<td>1.4</td>
</tr>
<tr>
<td>Readout bandwidth (Hz)</td>
<td>15.1</td>
<td>522.7</td>
<td>218.1</td>
<td>643.4</td>
<td>189.8</td>
</tr>
<tr>
<td>No. of signals acquired</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Sensitivity encoding factor</td>
<td>3 (AP)</td>
<td>2 (RL)</td>
<td>2 (RL)</td>
<td>1.5 (RL)</td>
<td>3 (RL)</td>
</tr>
<tr>
<td>Acquisition time</td>
<td>1 min 25 sec</td>
<td>5 min 12 sec</td>
<td>5 min 0 sec</td>
<td>6 min 3 sec</td>
<td>2 min 29 sec</td>
</tr>
</tbody>
</table>

Table 2

7-T VW imaging Protocol Used at Our Institution

<table>
<thead>
<tr>
<th>Imaging Parameter</th>
<th>3D T1 MPTR TSE</th>
<th>DW Imaging</th>
<th>3D T1 MPTR TSE with Contrast Agent</th>
<th>Inflow SWI with Contrast Agent (3 TEs)</th>
<th>3D T2 FLAIR with Contrast Agent</th>
</tr>
</thead>
<tbody>
<tr>
<td>FOV (mm²)</td>
<td>250 × 250 × 190</td>
<td>220 × 220 × 123</td>
<td>250 × 250 × 190</td>
<td>190 × 190 × 190</td>
<td>200 × 250 × 190</td>
</tr>
<tr>
<td>Acquisition orientation</td>
<td>Sagittal</td>
<td>Transverse</td>
<td>Sagittal</td>
<td>Transverse</td>
<td>Sagittal</td>
</tr>
<tr>
<td>Acquisition spatial resolution (mm³)</td>
<td>0.8 × 0.8 × 0.8</td>
<td>1.5 × 1.5 × 1.5</td>
<td>0.8 × 0.8 × 0.8</td>
<td>0.4 × 0.5 × 0.6</td>
<td>0.8 × 0.8 × 0.4</td>
</tr>
<tr>
<td>Reconstructed spatial resolution (mm³)</td>
<td>0.49 × 0.49 × 0.49</td>
<td>1.5 × 1.5 × 1.5</td>
<td>0.49 × 0.49 × 0.49</td>
<td>0.4 × 0.4 × 0.3</td>
<td>0.49 × 0.49 × 0.49</td>
</tr>
<tr>
<td>TR/TE/TI (msec)</td>
<td>3952/37/1375</td>
<td>17659/57/-</td>
<td>3952/37/1375</td>
<td>21/2.3*/-</td>
<td>8000/300/2200</td>
</tr>
<tr>
<td>Flip angle (degrees)</td>
<td>120</td>
<td>120</td>
<td>120</td>
<td>30</td>
<td>90</td>
</tr>
<tr>
<td>Echo spacing (msec)</td>
<td>3.3</td>
<td>3.3</td>
<td>3.3</td>
<td>...</td>
<td>4.7</td>
</tr>
<tr>
<td>TSE factor</td>
<td>169 (including 10 start-ups)</td>
<td>...</td>
<td>169 (including 10 start-ups)</td>
<td>...</td>
<td>125 (including 1 start-up)</td>
</tr>
<tr>
<td>Oversampling factor</td>
<td>1</td>
<td>...</td>
<td>1</td>
<td>v</td>
<td>1</td>
</tr>
<tr>
<td>Readout bandwidth (Hz)</td>
<td>934.8</td>
<td>23.6</td>
<td>934.8</td>
<td>557.0</td>
<td>410.9</td>
</tr>
<tr>
<td>No. of signals acquired</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Sensitivity encoding factor</td>
<td>2 (AP) and 3 (RL)</td>
<td>2.5 (AP)</td>
<td>2 (AP) and 3 (RL)</td>
<td>2.5 (RL)</td>
<td>2.5 (AP) and 3 (RL)</td>
</tr>
<tr>
<td>Acquisition time</td>
<td>10 min 40 sec</td>
<td>6 min 10 sec</td>
<td>10 min 40 sec</td>
<td>9 min 18 sec</td>
<td>10 min 48 sec</td>
</tr>
<tr>
<td>Head coil</td>
<td>32 channel</td>
<td>32 channel</td>
<td>32 channel</td>
<td>32 channel</td>
<td>32 channel</td>
</tr>
</tbody>
</table>

Note.—AP = anteroposterior direction, DW = diffusion weighted, FLAIR = fluid-attenuated inversion recovery, MPTR = magnetization-prepared inversion-recovery TSE (48), RL = right-left direction, TE = echo time, TI = inversion time, TR = repetition time, TFE = turbo field echo, VRITA = volumetric isotropically reconstructed TSE acquisition (43).

* Delta TE = 7.2 msec for second and third echo time.

Institution, is to only acquire postcontrast images. In our experience, most VW lesions—even when not enhancing—can be detected on the postcontrast VW imaging sequence and it saves half the acquisition time relative to the combination of a pre- and postcontrast sequence. Still, studies are needed to compare this alternative approach (postcontrast only) with the classic approach (pre- and postcontrast). One of the major limitations of using a postcontrast VW imaging sequence only is that findings such as intraplaque hemorrhage and intracranial arterial dissections may be missed (51).

Other Necessary Sequences

Other, more “conventional” MR imaging sequences also add helpful information when used in combination...
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Figure 2: A, 3-T transverse TOF MR image (voxel size, 0.4 × 0.7 × 1.0 mm³) and, B, corresponding 3T transverse oblique precontrast T1-weighted VW image (voxel size, 0.5 × 0.5 × 1.0 mm³), both zoomed in on the left P1/P2 bifurcation of the posterior cerebral artery (arrow). On the VW image (B), the basal vein of Rosenthal can be seen coursing directly lateral to the PCA (arrowhead), which is not visible on the TOF MR image (A).

Figure 3: A, 3-T oblique (transverse/coronal) precontrast T1-weighted VW image (voxel size, 0.5 × 0.5 × 1.0 mm³) in a 29-year-old healthy volunteer. In this single section, most large intracranial arteries of the circle of Willis are (partially) visible, for example, the left A1 segment of the anterior cerebral artery and basilar artery (arrows). B, Zoomed image of the M1 segment of the left MCA. The VW (arrow) can barely be seen because of its close proximity to the brain parenchyma, which has comparable signal intensity. C, Reconstructed sagittal image (0.6 × 1.0 in-plane spatial resolution) of the left MCA (arrow). The quality and detail are marginally lower compared with the transverse oblique image because of the anisotropic voxel size.

with intracranial VW imaging. MR angiographic methods, such as contrast-enhanced MR angiography or time-of-flight (TOF) MR angiography, are particularly helpful to assess the arterial lumen and to identify the specific arteries to which the visualized VWs belong (Fig 2). Also, in case of a small FOV, these techniques can guide FOV planning centered on the circle of Willis or focused on a specific (stenotic) VW lesion. Contrast-enhanced MR angiography during first contrast agent passage clearly shows the arterial vasculature and is less sensitive to slow-flow artifacts compared with TOF MR angiography. Furthermore, with strong elongation of the arterial vasculature it is also less sensitive to a signal decrease of cranial-caudal flow, for example, in a strongly elongated MCA.

Nonetheless, TOF MR angiography is still a sensitive method to detect stenotic lesions and is the workhorse in most centers for the detection of vascular disease, including aneurysms of the intracranial vasculature (52). Because it is not dependent on first contrast agent passage, acquisition time can be invested to increase the spatial resolution.

Other pulse sequences that can be of additional value in VW imaging assessment depend on the specific clinical question and include (a) T2-weighted TSE sequence that can confirm the absence of a flow void in the arterial lumen in a patient with an arterial occlusion, (b) T1-weighted anatomic sequence for both assessment of normal anatomy and for use as precontrast sequence for (mainly tissue) enhancement, (c) T1-weighted fat-suppressed sequence that can depict a subintimal hematoma in patients with an arterial dissection that involves both the extracranial and intracranial segments, and (d) fluid-attenuated inversion recovery and diffusion-weighted imaging sequence for localizing white matter lesions and old and recent ischemia possibly associated with VW disease.

The full VW imaging protocol used at our institution, including key pulse sequence parameters, can be found in Tables 1 and 2.

Patient Preparation

Proper patient preparation plays an important role in the acquisition of high-quality intracranial VW images. The patient (or legal representative) needs to be informed about the MR imaging examination, and the MR imaging staff needs to assess if any possible contraindications for MR imaging (claustrophobia, contraindicated metal objects in or on the body, pregnancy) or for gadolinium-containing contrast agents (known allergic reaction to gadolinium-containing contrast agent, severely impaired renal function) exist. Also, because of the relatively long acquisition time of VW imaging sequences, imaging staff needs to assess whether the patient is clinically able to undergo
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Willis. When the area of interest lies predominantly in the posterior circulation, a more transverse oblique angulation can be used to include a longer trajectory of the basilar and vertebral arteries. On the other hand, when there is suspicion of a dissection or intraplaque hemorrhage, a precontrast VW imaging sequence can be added to a postcontrast-only protocol. Further, previous (MR imaging) examinations showing arterial disease (eg, stenoses) at conventional sequences need to be taken into account for both FOV planning and evaluation of progression.

The patient’s treatment status is another important clinical feature for VW imaging assessment. For instance, when a patient with cerebral vasculitis is treated with prednisolone, it is important to know when treatment has commenced relative to the time of the imaging examination: VW enhancement may be lower or have disappeared altogether. Also, in case of an ischemic infarct that has been treated with thrombolysis or intra-arterial treatment, there may be local VW enhancement present at the location of the previous occlusion (54,55). Finally, if a patient has undergone an MR imaging examination that included contrast agent administration less than 12 hours before VW imaging, there may be residual contrast enhancement on the subsequent precontrast VW images.

Common Indications
At our institution, the most common indication for our 3-T VW imaging protocol is a suspected cerebral vasculitis (either primary angiitis of the central nervous system or due to other causes). When this suspicion is raised by the clinician, a spectrum of symptoms is often present that can also indicate the presence of other vascular diseases, such as small vessel disease or large artery atherosclerosis with unstable plaques. VW imaging can assist in differentiating between these vascular diseases but may also be helpful during follow-up and evaluation of the received treatment. Other vascular diseases in which VW imaging may be indicated are the detection or evaluation (evolution) of large artery

Pre-assessment Case Preparation—What Do We Need to Know?

Clinical Information
For a complete assessment of intracranial VW images, several clinical aspects are important to know. In general, when requesting VW imaging the actual clinical status of the patient (neurologic status, ability to lie still for a prolonged period of time) should be reported to assess the a priori chance of acquiring images of sufficient diagnostic quality. Next, a specific clinical question is important for defining a patient-tailored VW imaging protocol. For instance, if the clinician is suspicious of disease in the anterior cerebral vasculature, the (small) FOV of the 3-T sequence needs to be placed in a more transverse orientation in line with the arteries of the anterior circulation part of the circle of Willis. When the area of interest lies predominantly in the posterior circulation, a more transverse oblique angulation can be used to include a longer trajectory of the basilar and vertebral arteries. On the other hand, when there is suspicion of a dissection or intraplaque hemorrhage, a precontrast VW imaging sequence can be added to a postcontrast-only protocol. Further, previous (MR imaging) examinations showing arterial disease (eg, stenoses) at conventional sequences need to be taken into account for both FOV planning and evaluation of progression.

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General Assessment

VW imaging assessment at our institution is generally performed on specialized workstations for diagnostic imaging with a high-resolution monitor and a picture archiving and communication system (IDSS; Sectra AB, Linköping, Sweden). When an intracranial arterial stenosis has been found at TOF MR angiography, VW imaging could entail a dedicated characterization of this lesion on VW images (8). In practice, this is a less common situation in our patient population, so we perform a complete search of the large intracranial vasculature, starting at the extracranial internal carotid and vertebral artery and then following the course of these arteries into the basilar artery and anterior, middle, and posterior cerebral artery branches, including the M1, (very) proximal M2, A1, (very) proximal A2, P1, and (very) proximal P2 segments. For all currently published VW imaging sequences, including our own sequences, voxel size exceeds the VW thickness of all arteries and will result in overestimation of wall thickness. Lesions are visible, though, as they generally have a higher intensity or thicker appearance than the adjacent, apparently normal VW. In our experience, most intracranial VW lesions are detectable when scrutinizing the source data of the VW imaging sequences, that is, when the images are acquired in a transverse (7 T) or transverse oblique plane (3 T) (43). However, multiplanar reformating—when applicable—may increase diagnostic confidence of some lesions, including lesions of the M1 segment of the MCA, which is one of the few circle of Willis vessels visualized solely parallel to its orientation in a transverse section (Fig 1).

Before using intracranial VW imaging as a diagnostic tool in patients, one should become acquainted with the normal appearance of the arterial wall on VW images (Figs 1, 3). The normal VW can be seen as a thin line surrounding the vessel lumen and is isointense to the brain parenchyma (white and gray matter have approximately the same signal intensity on VW images because sequence parameters are specifically optimized for the VW). VW imaging of healthy intracranial vasculature frequently suggests a variance in wall thickness that is most likely not related to VW disease but due to partial volume effects. With normal ageing, the VW has been shown to increase in thickness; however, the question remains whether this small increase in atherosclerosis with or without arterial remodeling, and less frequently Moyamoya vasculopathy, dissections, and cerebral aneurysms (all mostly for research purposes). When 3-T VW imaging does not show any abnormalities, when the clinician suspects subtle VW disease, or when all arteries of the circle of Willis need to be thoroughly assessed and/or screened, our 7-T protocol can be considered if this platform is available.

Systematic Approach of VW Assessment

General Assessment

VW imaging assessment at our institution is generally performed on specialized workstations for diagnostic imaging with a high-resolution monitor and a picture archiving and communication system (IDSS; Sectra AB, Linköping, Sweden). When an intracranial arterial stenosis has been found at TOF MR angiography, VW imaging could entail a dedicated characterization of this lesion on VW images (8). In practice, this is a less common situation in our patient population, so we perform a complete search of the large intracranial vasculature, starting at the extracranial internal carotid and vertebral artery and then following the course of these arteries into the basilar artery and anterior, middle, and posterior cerebral artery branches, including the M1, (very) proximal M2, A1, (very) proximal A2, P1, and (very) proximal P2 segments. For all currently published VW imaging sequences, including our own sequences, voxel size exceeds the VW thickness of all arteries and will result in overestimation of wall thickness. Lesions are visible, though, as they generally have a higher intensity or thicker appearance than the adjacent, apparently normal VW. In our experience, most intracranial VW lesions are detectable when scrutinizing the source data of the VW imaging sequences, that is, when the images are acquired in a transverse (7 T) or transverse oblique plane (3 T) (43). However, multiplanar reformating—when applicable—may increase diagnostic confidence of some lesions, including lesions of the M1 segment of the MCA, which is one of the few circle of Willis vessels visualized solely parallel to its orientation in a transverse section (Fig 1).

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Table 3

| Commonly Seen Imaging Characteristics for Different Intracranial VW Diseases |
|---------------------------------|-----------------|-----------------|---------------------|--------------------------|--------------------------|
| VW Disease                      | Stenosis at MR Angiography/CT | VW Thickening | Location | Enhancement* | Special Considerations | Image Example |
| Intracranial atherosclerosis (35,42,47,56,70,72) | Equally present and not present | Eccentric | More widespread, distal ICA/vertebral, focal lesions | Equally present and not present | Plaque characterization, intraplaque hemorrhage† | Figure 4 |
| CNS vasculitis (73,74)          | Often present | Concentric | More widespread, long trajectory | Virtually always present | Effect of steroid therapy | Figure 9 |
| Moyamoya disease (75–79)        | Equally present and not present | Concentric | Distal ICA, proximal MCA | Equally present and not present | … | Figure 10 |
| Arterial dissection (80–83)     | Virtually always present | Eccentric | Distal ICA/vertebral | Often present | Detection of hematoma and flap, added value of fat suppression in case of an extracranial dissection | Figure 11 |
| Intracranial aneurysm (69,84–87) | Equally present and not present | … | … | … | … | … |
| RCVS (73,74,88)                 | Often present | Concentric | More widespread | Often not present | String-of-beads on angiograms | Figure 8 |
| Iatrogenic (after thrombectomy) (54,55) | Equally present and not present | Eccentric/concentric | Thrombectomy site | Often present | … | Figure 12 |

Note.—CNS = central nervous system, ICA = internal carotid artery, RCVS = reversible cerebral vasculopathy syndrome, SAH = subarachnoid hemorrhage.

† Concordant with contrast enhancement of extravascular disease, contrast enhancement of intracranial VW disease could reflect a more active phase of disease (74).

‡ Plaque characterization has mainly been performed in the Asian population, in which large multicomponent atherosclerotic plaques are significantly more prevalent. Several case series have shown the possibility of detecting intraplaque hemorrhage (22,89–93); however, postmortem studies suggest that intraplaque hemorrhage has a lower prevalence in intracranial plaques compared with extracranial (carotid) plaques (6,47,94); therefore, its role so far remains elusive.
thickness can be visualized with the currently used spatial resolutions (45). In younger patients without significant brain atrophy, the MCA is often located directly adjacent to the brain parenchyma, with little or no visible CSF in between. Because its signal intensity is similar to that of brain parenchyma in most sequences, it is often difficult to assess all segments of the MCA VW (including possible lesions) (Fig 3).

In general, a VW lesion is defined by one or both of the following characteristics (Fig 4): (a) a focal or more diffuse thickening of the VW greater than 30% compared with the adjacent VW thickness (31) and/or (b) focal or diffuse vivid contrast enhancement (56). One can further characterize the lesion as eccentric—less than 50% of the circumference of the VW—or concentric—greater than 50% of the circumference—and as enhancing or not enhancing after contrast agent administration (see below), which can give an indication of the specific underlying disease process (Table 3). When assessing a potential lesion, the area of interest should be compared with next sections (in case of concentric thickening) or the cross-section of the VW should be scrutinized (in case of eccentric thickening), and it should be compared with the contralateral VW (but beware of bilateral disease). Finally, VW images should be cross-correlated with the axial TOF MR angiographic images for correct interpretation of the specific artery/arteries in which the lesion(s) was/were found and to check if the lesion causes luminal narrowing. Bear in mind that due to arterial remodeling, VW lesions very often do not show a stenosis (15); therefore, the absence of stenosis should not be an argument in the decision process whether a lesion is present or not.

**Contrast Enhancement**

Ideally, both pre- and postcontrast images are available for assessment of VW lesion enhancement. With this setup, contrast-enhancing lesions can be distinguished more accurately from nonenhancing lesions with a high signal intensity. To this end, we either compare pre- and postcontrast images one-on-one (eyeballing) or we calculate subtraction images (postcontrast minus precontrast), for example, using MeVisLab (version 2.5; MeVis Medical Solutions, Bremen, Germany). However, as mentioned before, time constraints often limit VW imaging in clinical practice to one postcontrast VW imaging sequence. When only postcontrast images are available, assessment of contrast enhancement will be based on a comparison of the relative signal intensity of the arterial VW (lesion) with the appearance of the other (contralateral) arterial VW, the signal of the brain tissue, and/or the pituitary stalk. The pituitary stalk shows vivid contrast enhancement (57), and when the signal intensity of an intracranial arterial VW lesion approximates that of the pituitary stalk, it can be considered as contrast enhancement (Fig 4). Contrarily, if the signal intensity follows the intensity of the brain parenchyma, this can be considered as absence of enhancement.

In healthy subjects, contrast enhancement of the internal carotid artery and vertebral artery wall can be seen at the location where these arteries cross the dura mater and should not be mistaken for pathologic contrast enhancement (Fig 5). Vasa vasorum and increased permeability of the endothelium have been described at this location, but the exact nature of this contrast enhancement has yet to be determined (58). Also, the cavernous sinus shows diffuse enhancement after contrast agent administration; since the internal carotid artery runs through this cavernous venous plexus, it is difficult to assess the presence or absence of contrast enhancement of
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this cavernous segment of the internal carotid artery (Fig 5).

Common Artifacts and Corresponding Pitfalls

Like all MR imaging examinations, VW imaging is sensitive to motion artifacts, even more so because of the relatively long acquisition times. Also, sensitivity encoding fold-over artifacts need to be considered when deciding to use a VW imaging sequence with sensitivity encoding acceleration. Next to these more general artifacts, there are two artifacts that may pose an interpretation problem specifically when assessing VW images. The first is the slow-flow artifact (Fig 6). Due to the parabolic flow velocity profile within a vessel, the flow directly next to the VW is slower compared with the center of the lumen. Intracranial VW imaging sequences that use the inflow or outflow of blood to suppress the arterial lumen can therefore show a higher signal from unsuppressed blood near the VW, which mimics either the VW itself (making it appear thickened) or mimics a focal VW lesion (when flow is focally decreased). These hyperintense artifacts can be present both before and after contrast agent administration but are in principle more visible after contrast agent injection (Fig 6).

Slow-flow artifacts are most obvious in the slow-flowing blood in smaller and larger veins, for instance in veins within the Sylvian cistern that are in relatively close proximity to the branches of the MCA. When solely interpreting VW images, slow flow in veins can be misinterpreted as arterial wall enhancement. This misinterpretation can be avoided by routinely cross-checking the location of the intracranial arteries on TOF MR angiographic images or by assessing the pattern of the hyperintense signal: Slow-flow artifacts are often symmetrically present in both hemispheres and can be seen in many vascular structures both inside the skull and in the extracranial veins, such as the superficial temporal vein (Fig 6). Suboptimal suppression of slow flow is related to the VW imaging technique used, and although TOF MR angiography is also
prone to slow-flow artifacts, it rarely occurs that these artifacts occur at the exact same location at both sequences. When in doubt about whether a VW abnormality is a slow-flow artifact or a real VW lesion, we do not want to judge every abnormality as a VW lesion, but we analyse every possible lesion with caution; when in doubt, we may not call it a VW lesion; when the hyperintense signal is too large in volume for a thin arterial VW, we consider it a slow-flow artifact.

The other artifact that can cause difficulties in interpretation of VW abnormalities is the free-induction decay artifact (Fig 7). This artifact is caused by repeated refocusing radiofrequency pulses within the short echo times of T1-weighted VW imaging sequences. The artifact occurs when the free-induction decays from the refocusing pulses are not completely spoiled and appears as a linear dashed pattern on the image (59). The VW will show the same dashed, “zigzag” pattern, making it difficult to differentiate between artifact and VW thickening and/or lesion. Possible ways to minimize this artifact is to increase the number of signals acquired, increase the echo time, or choose a larger section thickness; however, for obvious reasons these are difficult to implement in intracranial VW imaging.

VW Disease

Although intracranial VW imaging is still in its developmental stage, there are already several challenging diagnostic situations in which it has shown its potential value (1,2,60,61), the most important of which is determining the cause of stroke and assessing accompanying VW lesions. Recently, two excellent review papers have been published that both address the commonly found imaging characteristics of different types of intracranial VW disease, including a thorough literature overview (2,62). We will therefore restrict this section to a compact overview table (Table 3) for hands-on use in clinical practice, including illustrative image examples (Figs 4, 8–12).
Future Prospects

Technical Developments

As discussed in the previous paragraphs, the difficulty in intracranial VW imaging is that ideally, one strives for a maximum spatial resolution to detect lesions of the small intracranial arterial VWs and maximum suppression of both blood signal within and CSF signal surrounding the arteries. Both will significantly increase acquisition time. For clinical implementation, acquisition time reduction is necessary to perform both pre- and postcontrast VW imaging within a clinical MR imaging time slot (approximately 25–35 minutes), without a substantial decrease in SNR and CNR of the arterial VW. Innovative methods for reducing acquisition time (eg, compressed sensing [63,64]), as well as different CSF suppression techniques (DANTE [19,20,65], antidriven-equilibrium [32,33]), in combination with various 2D and 3D acquisition methods with different coverage, need to be compared in the upcoming years.

Spatial coverage.—At 3 T, most sequences are limited to a region that covers either the circle of Willis or the (known) stenotic VW lesion, with a few surrounding centimeters. An option for “increasing coverage” is to acquire the VW images in a more angulated coronal plane and include the proximal vertebral arteries, as we generally do with our 3-T sequence. “Real” whole-brain coverage—that is, increasing the FOV—benefits from nonselective pulses (shorter echo spacing in TSE trains), 2D sensitivity encoding, and no need for oversampling (48). However, because flow suppression is often based on dephasing during the echo train, with too short echo train lengths flow suppression will inevitably decrease.

Next to improvements in pulse sequence design, hardware improvements may also show promise in VW imaging. Recently, an advanced coil system for joint intracranial and extracranial VW imaging has been developed (66). This coil system provides the opportunity to image both intracranial and extracranial arteries at once for an optimal assessment of the association of carotid and intracranial atherosclerotic plaques and ischemic stroke within a reasonable imaging time (5 minutes 54 seconds to 7 minutes 36 seconds).

Acquisition time.—Efficient k-space sampling trajectories (view-ordering) in combination with parallel imaging techniques have been described to
reduce acquisition time in 3D TSE sequences, while the trajectories can also be optimized for reduced T2 weighting (17,23). Compressed sensing allows image reconstruction from fewer k-space data and, thus, shorter imaging times. Compressed sensing needs yet to be investigated for intracranial VW imaging but might be a way to further reduce the acquisition time (63,64).

**Clinical Considerations**

Because no standard of reference in vivo method for intracranial arterial VW disease is available, histologic validation of intracranial VW imaging is essential. Since no tissue can be obtained while the patient is alive—compared with, for example, endarterectomy samples in carotid artery disease—validation can only be performed in postmortem studies. A series of postmortem validation studies has been performed at 7 T using ex vivo circle of Willis specimens from patients with and without a history of cerebrovascular disease, as well as from patients with intracranial aneurysms (47,67–71). These studies found clear correlations between VW and atherosclerotic plaques detected on VW images and histopathologic findings, best seen on T1-weighted data.

In current protocols, several imaging parameters can be varied to counterbalance the relatively long acquisition time, such as the oversample factor, TSE train length, and image acceleration by parallel imaging such as sensitivity encoding that leads to a change in imaging time (1,21,31,40). However, all of these techniques sacrifice spatial resolution and/or SNR in the process or need more advanced hardware (eg, a higher number of receiver coils or multiband imaging) to overcome the inevitable cost in image quality.

**Figure 11**

A–C, 7-T transverse VW images in a 44-year-old woman with a spontaneous dissection of the left ICA, imaged 4 days after symptom onset. A, B, Precontrast (A) and postcontrast (B) T1-weighted magnetization-prepared inversion-recovery TSE images show a tapering of the lumen and VW enhancement (arrow) of the left distal ICA. C, Diffuse concentric VW enhancement and a dissection flap are seen on the postcontrast image more proximally in the left distal ICA at the skull base (arrow). D, Tapering of the distal ICA can also be appreciated on a CT angiogram, reformatted in the same orientation as, A, and, B.

**Figure 12**

7-T precontrast sagittal (A) and coronal (B) T1-weighted magnetization-prepared inversion-recovery TSE images of an anterior communicating artery aneurysm. The aneurysm wall shows a variation in wall thickness: The left lateral side and top of the aneurysm can be seen to have a thicker wall (white and black arrowheads), while the right lateral and anterior part of the aneurysm show a thinner wall (white and black arrows). C, 7-T maximum intensity projection of 3D TOF MR angiography shows the corresponding anatomic geometry of the aneurysm (arrowheads).
sequences (47,67,68,70,71). However, more insight into what normal ageing of the VW looks like on VW images and what underlying mechanisms can cause VW (lesion) enhancement are still needed. This has proven to be a challenge, because methods to preserve tissue in ex vivo studies (eg, fixation and tissue temperature effects) change the MR imaging characteristics of the tissue so that results from these studies cannot be directly translated to in vivo VW imaging, while functional measures, such as lesion enhancement after contrast agent administration, cannot be performed in postmortem studies.

**Intracranial VW imaging has become part of state-of-the-art MR imaging protocols detecting causes of ischemic stroke, mainly in a research setting but increasingly asked for (and used) in clinical practice. It has tentatively shown commonly seen VW changes in patients with diseases including, but not limited to, central nervous system vasculitis, Moyamoya disease, aneurysms, dissections, and intracranial atherosclerosis. However, its precise role and added value for prognosis and patient care needs further elucidation. A field strength of at least 3 T enables VW imaging sequences with high enough CNR and spatial resolution to assess the thin intracranial atherosclerotic VW and detect and ultimately characterize VW lesions. Radiologists should be aware of the normal appearance (variance) of the VW on intracranial VW images, the main characteristics of VW lesions that may help differentiate between different VW pathologic conditions, and the technical limitations and pitfalls in the assessment of intracranial VW imaging. Further technical improvements will enable (among others) reduced acquisition time, while further histologic validation of these VW imaging sequences will aid in a better understanding of normal VW thickness variance, ultimately leading to better knowledge of the underlying pathologic conditions of lesions seen on VW images.**

**Acknowledgments:** The authors acknowledge Chris van Kesteren and Roy Sanders for their image drawings of Figure 1 and Anita Hargetveld, PhD, and Nikki Dieleman, PhD (Department of Radiology) and Rachel Kleinbloog, MD (Department of Neurology), the UMC Utrecht, the Netherlands, for providing patient data for Figures 5, C and D, 9, and 12.

**Disclosures of Conflicts of Interest:** A.L. Activities related to the present article: grant from European Research Council (ERC:2014-STG - 637024, HEARTOFSTROKE). Activities not related to the present article: disclosed no relevant relationships. Other relationships: disclosed no relevant relationships. A.G.v.d.K., disclosed no relevant relationships. J.J.M.Z., disclosed no relevant relationships. J.H. Activities related to the present article: disclosed no relevant relationships. Activities not related to the present article: starting grant from European Research Council (no. 337333). Other relationships: disclosed no relevant relationships.

**Conclusion**

Intracranial VW imaging has become part of state-of-the-art MR imaging protocols detecting causes of ischemic stroke, mainly in a research setting but increasingly asked for (and used) in clinical practice. It has tentatively shown commonly seen VW changes in patients with diseases including, but not limited to, central nervous system vasculitis, Moyamoya disease, aneurysms, dissections, and intracranial atherosclerosis. However, its precise role and added value for prognosis and patient care needs further elucidation. A field strength of at least 3 T enables VW imaging sequences with high enough CNR and spatial resolution to assess the thin intracranial atherosclerotic VW and detect and ultimately characterize VW lesions. Radiologists should be aware of the normal appearance (variance) of the VW on intracranial VW images, the main characteristics of VW lesions that may help differentiate between different VW pathologic conditions, and the technical limitations and pitfalls in the assessment of intracranial VW imaging. Further technical improvements will enable (among others) reduced acquisition time, while further histologic validation of these VW imaging sequences will aid in a better understanding of normal VW thickness variance, ultimately leading to better knowledge of the underlying pathologic conditions of lesions seen on VW images.

**References**


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60. Kontzialis M, Wasserman BA. Intracranial vessel wall imaging: current applications.


**QUESTIONNAIRE**  
A13(20)  
The use and pitfalls of intracranial vessel wall imaging: how we do it

**INSTRUCTIONS**
- Read through the article and answer the multiple-choice questions provided below.
- Some questions may have more than one correct answer; in which case you must please mark all the correct answers.

<table>
<thead>
<tr>
<th>Question 1: Which one of the following is the leading cause of ischemic stroke worldwide?</th>
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<tr>
<td>A: Vasculitis after bacterial meningitis</td>
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<td>B: Atherosclerotic plaques in carotid arteries</td>
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<tr>
<td>C: Intracranial atherosclerosis</td>
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<td>D: Uncontrolled epilepsy</td>
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<th>Question 2: Which one of the following is a major advantage of intracranial vessel wall (VW) imaging over lumen-based methods?</th>
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<tr>
<td>A: It can depict stenotic lesions in large, as well as small arteries</td>
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<td>B: It can detect arterial VW calcifications that are associated with future stroke risk</td>
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<td>C: It is an easily accessible and fast procedure in patients with acute stroke</td>
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<td>D: It can further characterize stenotic lesions that have already been detected with common angiographic methods</td>
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<tr>
<th>Question 3: Which one of the following is TRUE regarding the contrast-to-noise ratio (CNR) in intracranial VW imaging?</th>
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<tr>
<td>A: A high CNR and a low spatial resolution are needed to visualize the thin arterial VW and to characterize VW lesions</td>
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<td>B: CNR in VW imaging comprises signal contrast of the VW relative to its direct surroundings, that is, brain tissue and cerebrospinal fluid (CSF)</td>
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<td>C: The first step in achieving a high VW CNR is to enhance the luminal blood</td>
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<td>D: At 3 T, the CNR between VW and lumen is superior to the CNR between VW and CSF on most pulse sequences</td>
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<th>Question 4: Which one of the following is TRUE regarding two-dimensional (2D) versus 3D imaging?</th>
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<tbody>
<tr>
<td>A: The 2D in-plane spatial resolution is higher than that of 3D</td>
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<td>B: The drawbacks of 3D spin-echo methods include the need to target a specific lesion due to limited coverage</td>
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<td>C: 2D spin-echo sequences can be used as an alternative to 3D methods to increase in-plane spatial resolution</td>
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<tr>
<td>D: 2D methods can not be used to characterize the underlying pathologic process of a lesion</td>
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<th>Question 5: Which one of the following is FALSE regarding the use of contrast agents?</th>
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<tr>
<td>A: Sensitivity for contrast enhancement depends on specific patient characteristics</td>
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<td>B: No head-to-head comparisons between VW imaging sequences at different time points after contrast agent injection have been performed recently</td>
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<td>C: Optimal timing of sequence acquisition is approximately 5–10 minutes after contrast agent injection</td>
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<td>D: The choice of whether or not to obtain both pre- and postcontrast images depends on both the specific setting and clinical question</td>
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<th>Question 6: Which one of the following is a particularly helpful use of contrast enhanced MR angiography?</th>
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<td>A: Identifying slow-flow artifacts</td>
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<td>B: Assessing the arterial lumen</td>
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<td>C: Identifying vessels at risk for vasculitis</td>
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<td>D: Assessing lesions in the venous lumen</td>
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<th>Question 7: Which one of the following is important to ask before intracranial VW imaging?</th>
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<td>A: What is the patient’s weight and BMI?</td>
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<td>B: Does the patient have a family history of cancer?</td>
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<td>C: Is the patient allergic to bees?</td>
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<td>D: What is the patient’s current treatment status?</td>
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<th>Question 8: What is the MOST common indication for the 3-T VW imaging protocol at the institution used in this study?</th>
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<td>A: Suspected cerebral vasculitis</td>
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<td>B: Signs of raised intracranial pressure</td>
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<tr>
<td>C: Family history of brain tumors</td>
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<td>D: Suspected intracranial hemorrhage</td>
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<th>Question 9: Which one of the following is TRUE regarding the commonly seen imaging characteristics of different intracranial VW diseases?</th>
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<td>A: String-of-beads on angiograms is suggestive of arterial dissection</td>
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<td>B: Moyamoya disease presents with eccentric VW thickening</td>
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<td>C: In CNS vasculitis enhancement is virtually always present</td>
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<td>D: Iatrogenic vasculitis often enhances when symptomatic, but is difficult to assess when SAH is present</td>
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</table>
**Question 10:** Is it TRUE or FALSE that this study recommends a systematic approach to searching for lesions of the large intracranial vasculature, starting at the basilar artery and anterior, middle, and posterior cerebral artery branches and then following the course to the extracranial internal carotid and vertebral artery?

A: TRUE  
B: FALSE

**Question 11:** Which one of the following is TRUE regarding the definition and characterization of a lesion?

A: A lesion is defined as a focal thickening of the VW less than 50% compared to the adjacent VW thickness  
B: A lesion is defined as a focal or diffuse vivid contrast enhancement  
C: One can characterize a lesion as concentric if less than 50% of the circumference of the VW is affected  
D: Contrast enhancement gives no indication of the specific underlying disease process

**Question 12:** Which one of the following is TRUE regarding slow flow artifacts?

A: Slow flow artifacts can be present both before and after contrast agent administration, but are usually more visible before contrast agent injection  
B: Slow-flow artifacts are most obvious in the high-pressure blood in smaller arteries  
C: Slow-flow artifacts are often symmetrically present in both hemispheres and can be seen in many vascular structures both inside the skull and in the extracranial veins  
D: Slow-flow artifacts are caused by repeated refocusing radiofrequency pulses within the short echo times of T1-weighted VW imaging sequences

**Question 13:** How can free-induction decay artifact be minimized?

A: Decreasing the number of signals acquired  
B: Increasing the amount of contrast material  
C: Decreasing the echo time  
D: Choosing a larger section thickness

**Question 14:** Is it TRUE or FALSE that innovative methods for reducing acquisition time, like compressed sensing, as well as different CSF suppression techniques in combination with various 2D and 3D acquisition methods with different coverage, have been used in the past but have been discarded due to increased financial burden?

A: TRUE  
B: FALSE

**Question 15:** Is it TRUE or FALSE that further improvements in histologic validation of VW imaging sequences will aid in a better understanding of normal VW thickness variance, ultimately leading to better knowledge of the underlying pathologic conditions of lesions seen on VW images?

A: TRUE  
B: FALSE

END
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(If your personal details have not changed, only complete the sections marked with an asterisk *)

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Circle your specialty
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- CT
- MRI
- X Ray
- Mammography
- Ultrasound
- Radiation Oncology
- Nuclear Medicine

ANSWER SHEET
A13 (20)
The use and pitfalls of intracranial vessel wall imaging: how we do it

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Signed: Date:

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