Diagnosis of stroke with advanced CT and MR imaging

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There have been important advances in stroke imaging, including CT perfusion imaging, xenon-CT, CT angiography, MR diffusion imaging, MR perfusion imaging, MR angiography and haemorrhage-sensitive gradient echo MR sequences.

The technical principles and clinical applications of these methods are explained. An emphasis is made on the diagnosis of hyperacute cerebral ischaemia and issues surrounding the differentiation of reversible from irreversible ischaemic damage with modern imaging modalities, which has implications for thrombolytic therapy. This is followed by an overview of the role of imaging in patients with chronic stroke and transient ischaemic attack. In these patients, the diagnostic contribution of MRI in detecting the underlying pathology and the assessment of cerebrovascular reserve with perfusion imaging form an important part in the secondary prevention of stroke.

Technical advances in cross-sectional imaging

Computed tomography (CT) of the brain has been the mainstay of imaging patients with an acute neurological deficit. In recent years, magnetic resonance imaging (MRI) has been increasingly used in addition to CT. There have been important technological advances in both modalities. This development has been driven to a large extent by the need for early stroke detection prompted by the fact that active treatment with thrombolytic agents was beneficial and is beginning to be more widely used. An overview of technical advances will precede a discussion of the imaging in acute ischaemia, which is the main focus of this chapter, followed by considerations pertaining to the imaging of subacute and chronic cerebral ischaemia.

Advances in CT scanning

Spiral CT scanning
All modern CT scanners provide now a facility for spiral CT scanning. In conventional incremental CT scanning, slices are acquired individually
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with a time delay between them for table repositioning and tube cooling. Spiral (or helical) scanning is performed with a continuous rotation of the X-ray tube during simultaneous movement of the CT table at a pre-selected speed. This allows the acquisition of a volumetric 3D data set, which can subsequently be divided into individual slices of variable thickness¹. Spiral scanning provides a considerable increase in scanning speed and allows coverage of the entire head in approximately 15 s. This can be of advantage in restless patients with acute stroke and represents a prerequisite for CT angiography where imaging is performed during the relatively short intravascular phase of a contrast medium (see below). The most recent developments in this field are multi-slice spiral CT scanners, which use multiple detectors for simultaneous data collection at different locations and can acquire images at 3 times the speed of single detector spiral CT scanners².

CT perfusion imaging

There are two fundamentally different methods by which CT scanning can be used to assess cerebral perfusion. One uses the inhalation of xenon, the other a bolus injection of an iodinated contrast medium³⁴.

Xenon is a stable gas, which has an atomic number close to iodine and, therefore, attenuates the X-ray beam in a similar fashion. Unlike iodine, xenon is freely diffusable and penetrates the blood–brain barrier. Current set-ups for xenon CT scanning consist of the inhalation of a gas containing 28% xenon during sequential acquisition of CT images over a period of approximately 6 min. The distribution of xenon in the brain depends on the regional blood flow and is slightly quicker in grey matter than in white matter. The change of the Hounsfield numbers (CT numbers) over time during inhalation of xenon forms the basis of blood flow calculations, which are usually displayed as colour maps. The wash-out of xenon occurs relatively rapidly, allowing a repeat examination after 15–20 min. A disadvantage of this method is that any patient movement during the 6 min period causes misregistration of data. Xenon uptake may also be impaired in patients with severe pulmonary disease.

The second technique of CT perfusion imaging, tracks transients changes in the blood vessels and brain parenchyma during the first pass passage of an intravenously injected contrast medium, similar to MR perfusion imaging (see below). A series of images is acquired at a predetermined level with a temporal resolution of one image every 1 or 2 s. The passage of the contrast-medium bolus causes a transient increase in Hounsfield units, which is proportional to the iodine concentration in the perfused tissue. Maps of cerebral blood volume (CBV), mean transit time (MTT) and cerebral blood flow (CBF) can be obtained from a pixel-by-pixel analysis of the density changes over time.
Absolute quantification of cerebral blood flow is theoretically possible with this method because of the linear relationship between iodine concentration and Hounsfield numbers, but there remains some doubt about its accuracy in practice. Blood flow measurements of cortical grey matter using the bolus perfusion technique were systematically lower compared to data from xenon CT studies.

CT angiography
Selective imaging of blood vessels with CT has become possible with the introduction of spiral CT scanners. The image quality of CT angiography is likely to improve dramatically with the advent of the new multi-slice spiral CT scanners. Data are acquired during the vascular phase of an iodinated contrast, which is injected intravenously, typically at a rate of 3 ml/s. The enhanced blood vessels are extracted from 3D data sets by applying specific density thresholds during the post-processing. The vessels can then be displayed as 2D projectional images, which resemble conventional angiograms, or as 3D surface rendered structures. CT angiography can be performed rapidly and can be easily ‘tagged onto’ a routine diagnostic CT. It has the disadvantage of using iodinated contrast medium, which carries a small risk of adverse reactions. It may also be difficult to isolate blood vessels running close to bone during post processing.

Advances in MR imaging

MR perfusion imaging
MR perfusion imaging exploits magnetic susceptibility effects within the brain tissue during the first pass of an intravenously injected gadolinium-based contrast agent. During its first pass through the brain, the contrast medium causes a transient signal drop on $T_2^*$-weighted (susceptibility-weighted) MR images (Fig. 1). Images are typically acquired with a temporal resolution of one image every 1–2 s, similar to CT perfusion. The use of single-shot echoplanar imaging (EPI), however, allows multislice imaging with full brain coverage, which represents a distinct advantage compared to the single slice technique in CT perfusion imaging. MR perfusion imaging is, however, at present only semiquantitative and cannot provide absolute values. The sequential changes in signal intensity can by plotted as a time-signal intensity curve of a chosen region of interest or reproduced as pixel based colour maps (Fig. 1). The relative cerebral blood volume (rCBV) is proportional to the area under the curve on the time-signal intensity graph. Other measurements that can be derived are arrival time ($T_0$) and mean transit time (MTT) of the gadolinium bolus. Using tracer kinetics, the relative cerebral blood flow (rCBF) can be estimated by dividing the relative blood volume by the
Fig. 1 MR perfusion study in a patient with bilateral carotid artery occlusion. An MRA of the intracranial circulation (a) does not demonstrate any internal carotid arteries. Both middle meningeal arteries (arrows) are prominent and provide collateral supply to the brain. The right posterior cerebral artery (curved arrow) is normal but only the proximal part of the left posterior cerebral artery is seen. T2* weighted EPI images before (b) and 20 s after injection of a gadolinium bolus (c) demonstrate the magnetic susceptibility effects of the passing gadolinium bolus, which leads to a decrease in signal intensity of the blood vessels and brain parenchyma in (c) compared to (b). The time course of this signal change is shown in a time-signal intensity graph (d) for two separate regions of interest: the right occipital lobe (region 1) and left frontal lobe (region 2). The signal drop in the right occipital occurs much earlier and is more marked than in the left frontal region, where perfusion is impaired.

A colour map (e) of the mean transit time (MTT) shows areas with a prolonged transit time in green and red. Prolongation of the MTT, which corresponds to a delayed passage of the bolus, is found in watershed areas at the boundaries between the anterior and middle and between the middle and posterior cerebral arteries.
Fig. 2 Diffusion-weighted and T2 spin-echo images in a patient with a right subcortical infarct. Although the lesion is seen on the T$_2$-weighted image (a), it is much more conspicuous on the diffusion-weighted image (b), which confirms that the lesion is acute. The diffusion of water molecules is restricted in an area of acute ischaemia, which appears therefore a bright on diffusion-weighted images ("light bulb sign") Note that the CSF, which has a high degree of molecular motion, appears dark.

mean transit time ($r$CBF = $r$CBV/MTT)$^8$. In the absence of absolute quantification of the cerebral blood flow, comparison with the contralateral hemisphere provides the easiest way to analyse MR perfusion images. However, this becomes problematic if the perfusion of the contralateral hemisphere is not normal, as in the presence of bilateral carotid artery disease.

**MR diffusion imaging**

Diffusion-weighted MR imaging exploits the presence of random motion (Brownian motion) of water molecules to produce image contrast, thereby providing information not available on standard T$_1$- or T$_2$-weighted images$^9$. This is achieved by applying a pair of ‘diffusion’ gradients symmetrically around a 180° refocusing radio frequency (RF) pulse of a T$_2$-weighted MR sequence. Mobile molecules acquire phase shifts, which prevent their complete rephasing and result in signal loss. The loss of signal is proportional to the degree of microscopic motion that occurs during the pulse sequence. On diffusion-weighted images, regions of relatively stationary water molecules appear much brighter than areas with a higher molecular diffusion (Fig. 2). The degree of phase shift and signal loss depends also on the strength and duration of the ‘diffusion’
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gradient, which is expressed by the ‘b value’. B-values used for imaging of acute stroke lie typically around 1000 s/mm$^2$. Quantitative analysis of the apparent diffusion coefficient (ADC) requires scanning with at least two different b-values and additional postprocessing. ADC maps are solely based on differences of tissue diffusion, independent of any T$_2$ effects$^9$. The ADC in the normal brain ranges from 2.94 x 10$^{-3}$ mm$^2$/s for CSF to 0.22 x 10$^{-3}$ mm$^2$/s for white matter; grey matter lying in between with a ADC of 0.76 x 10$^{-3}$ mm$^2$/s$^{10}$. Areas with a decreased ADC appear dark on ADC maps, which is the converse to diffusion-weighted images where areas of decreased diffusion appear bright$^9$.

MR angiography
MR angiography can be performed at the same time as MR imaging of the brain parenchyma and forms an important part in the work-up of acute and chronic stroke. Its technical principles and applications are discussed by Clifton elsewhere in this issue.

Imaging of acute stroke

Pathophysiological considerations
The pathophysiology of acute stroke represents a chain of rather complex events, which can be simplified into three consecutive stages for the purpose of explaining the evolution of imaging findings: (i) flow abnormalities; (ii) cellular dysfunction; and (iii) structural breakdown$^{11,12}$. Flow abnormalities represent a kinetic phenomenon, which can be detected immediately after the onset of stroke, both at the macrovascular and at microvascular level; the former by MRA and CTA, the latter by CT perfusion and MR perfusion imaging.

The second stage, cellular dysfunction, occurs if the cerebral blood flow falls below a critical level. At 20 ml/100 g/min, the electrical activity in the brain ceases and the water homeostasis begins to be disrupted. At 10–15 ml/100 mg, failure of the energy-dependent sodium pump leads to an accumulation of intracellular sodium and water moves into intracellular compartments causing swelling of neurons (cytotoxic oedema). Cytotoxic oedema predominates during the first 6 h following the onset of ischaemia. It is most conspicuous on diffusion-weighted MR images, but is also responsible for the early CT signs. Energy failure leads to an accumulation of lactate, which can be detected with spectroscopy (see Saunders elsewhere in this issue). As time proceeds, structural breakdown of the blood–brain barrier occurs with leakage of intravascular fluid and protein into the extracellular space (vasogenic oedema). The breakdown of the blood–brain barrier is a result of ischaemic damage to the
endothelial lining of the capillaries, which is less oxygen dependent than neurons. Vasogenic oedema therefore develops after 6 h, reaching its peak between 24–48 h after the onset of ischaemia. Vasogenic oedema causes brain swelling, produces low-attenuation on CT and high signal on T₂-weighted and fluid-attenuated inversion recovery (FLAIR) images.

**CT imaging of acute stroke**

**Exclusion of haemorrhage**

CT scanning has been the modality of choice for imaging patients with acute stroke. Its major advantage, apart from wide-spread accessibility, is its reliability in detecting intracerebral and subarachnoid haemorrhage. It is, therefore, highly sensitive in distinguishing haemorrhagic from non-haemorrhagic stroke. Rare exceptions are very anaemic patients with a haematocrit of 20% or less, in whom haematomas can be isointense. All major thrombolysis trial to date have used CT as primary imaging modality to exclude haemorrhage or extended infarction.

**Large vessel occlusion**

A ‘dense middle cerebral artery sign’ is the earliest detectable change on CT and can be seen at the onset of the ictus. It is the result of increased attenuation of the horizontal first segment of the middle cerebral artery, which contains thrombus. The presence of a ‘dense middle cerebral artery sign’ correlates with large infarcts and worse patient outcome. One pitfall in diagnosing a dense middle cerebral artery sign is the presence of heavily calcified intracranial arteries, but these are usually bilateral.

CT angiography appears promising in the detection of acute middle cerebral artery occlusion and can be combined with delayed phase imaging, which shows enhancement of the collateral circulation.

**CT perfusion imaging**

The effects of a proximal artery occlusion on the microvascular circulation can be assessed with CT perfusion. Xenon CT studies have been used to produce quantitative perfusion data in patients with angiographically proven acute MCA occlusions, 55% of whom had a normal CT: patients with a M1 segment occlusion had a mean CBF of 12 ml/100 g/min compared to 30 ml/100 g/min in patients without a M1 occlusion. Xenon CT also proved to be a useful predictor for the development of severe oedema with life-threatening brain herniation. This was observed in patients with a mean CBF of 8.6 ml/100 g/min compared to a mean CBF of 18 ml/100 g/min in patients with only mild oedema. Koenig used CT perfusion imaging following an intervenous bolus injection in patients...
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presenting within 6 h after onset of an acute stroke. He demonstrated a reduction of CBF in 25 of 28 patients who subsequently progressed to develop an infarct. The 3 patients missed with this technique developed the infarct outside the scanned level, which demonstrates the limitation of a single slice technique.

Early signs on conventional CT

Early signs of parenchymal change in acute ischaemia are obscuration of the lentiform nucleus, loss of the insular ribbon and loss of the differentiation between cortical grey and subcortical white matter. These changes reflect neurotoxic oedema with accumulation of water in grey matter structures, which decreases their Hounsfield number and makes them no longer distinguishable from the adjacent white matter. A change of 4 Hounsfield units or more is visually detectable and corresponds to a change in water content of approximately 1.5%. In cases of severe ischaemia with poor collateral circulation, cytotoxic oedema produces a 3% increase in water content within 1 h and a 6% increase within 4 h. Severe ischaemia can, therefore, be detectable on a CT performed at 1 h but areas with marginal cerebral blood flow between 15–20 ml/100 g/min may remain undetected on early CT. Later, vasogenic oedema causes effacement of the cerebral sulci and diffuse parenchymal hypodensity. Extensive diffuse hypodensity on early CT scans involving over 50% of the MCA territory is associated with a high mortality from malignant brain oedema.

It must be emphasised that early ischaemic changes on CT scans are subtle and may easily be overlooked by less experienced observers. The CT scans of patients entered into the European Cooperative Acute Stroke Study (ECASS) underwent a post hoc review by experts, which showed that the initial ‘on site’ interpretation overlooked an early infarct in 11% of the patients.

MR imaging of acute stroke

Exclusion of haemorrhage

Detection of hyperacute haemorrhage on conventional spin echo MR sequences is more difficult than on CT. The MR appearances of haemorrhage depend on the presence of paramagnetic haemoglobin breakdown products such as deoxyhaemoglobin, methaemoglobin and haemosiderin. Conventional spin echo sequences are sensitive to subacute and chronic haemorrhage, but the detection of hyperacute and acute haemorrhage can be greatly improved by using a T<sub>2</sub>*-weighted gradient echo sequence. It is much more susceptible to paramagnetic breakdown products, which cause signal loss and appear as areas of low intensity. Whether T<sub>2</sub>*-weighted GRE is as sensitive as CT in excluding haemorrhage remains to be proven.
Occlusion of large vessels

Occlusion of a major intracerebral blood vessel may be evident by a lack of flow void on conventional spin echo images or can be diagnosed with MRA (see Fig. 1). MRA is discussed in detail in a separate chapter. It is worth mentioning that, in the context of acute stroke, MRA has been shown to correlate well with conventional angiography in demonstrating vascular occlusion prior to and recanalisation following thrombolytic treatment.

MR perfusion imaging

MR perfusion imaging provides immediate and direct evidence of the repercussions of proximal vessel occlusion on the microcirculation. A prolongation of the mean transit time (MTT) is the earliest and most consistent sign of an impairment in perfusion. The relative cerebral blood volume (rCBV) may be normal or increased in the presence of adequate collateral supply, but is decreased if the collateral circulation is insufficient. The response of rCBV to a prolongation of MTT provides information about the autoregulatory capacity, which aims to maintain the cerebral blood flow (rCBF) by increasing rCBV (rCBF = MTT/rCBV) by vasodilatation and recruitment of collaterals.

Sorenson initially studied patients in the first 10 h and showed abnormalities on MTT maps to be more extensive than on rCBV maps. In a subsequent study, he performed rCBF measurements from MR perfusion studies and showed that, in the hyperacute state, blood flow maps revealed abnormalities not visible on blood volume maps, but that the final infarct in these untreated patients corresponded more closely to the blood volume maps. Ueda showed that the size of the final infarct was grossly over-estimated on MTT maps, but correlated well with the extent of abnormality shown on rCBV maps.

MR diffusion imaging

MR diffusion imaging is by far the most sensitive MR technique for the detection of early parenchymal change following an ischaemic insult. Water diffusion is restricted in areas of ischaemic damage which appear therefore bright on diffusion weighted images (‘light bulb sign’) or dark on ADC maps. The precise mechanisms leading to a reduction in diffusion are actively debated, but redistribution of extracellular water into the intracellular compartment (cytotoxic oedema), resulting in shrinkage of the extracellular space appears the most likely explanation. Diffusion-weighted imaging is highly sensitive and specific for the detection of early ischaemia and infarcts within the first 6 h of onset. MR diffusion imaging has, therefore, a pre-eminent role in the assessments of patients presenting within the therapeutic window for thrombolysis, as routine T₂ weighted images will be normal at this stage (Fig. 3). Diffusion-weighted
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Fig. 3 34-year-old patient with a locked-in syndrome. The frontal projection of an MRA of the posterior circulation (a) shows occlusion of the basilar artery distal to the right anterior inferior cerebellar artery (curved arrow) with filling of the posterior cerebral arteries through collaterals (arrows). The $T_2$-weighted image in the hyperacute phase (b) is normal but the diffusion-weighted image shows high signal in the pons (c), which is becomes visible on $T_2$-weighted image after 48 h (d).

Images may also be useful in differentiating between a transient ischaemic attack (TIA) and an infarct, since they appear to be normal in the former.

MRI diffusion imaging was also shown to be useful in subacute infarcts. In the presence of several abnormalities on $T_2$-weighted images, diffusion-weighted images can pinpoint the acute lesion and determine its vascular territory, which was felt to be clinically relevant in 48% of the cases in a recent clinical study.

Combined MR perfusion and diffusion imaging

Cerebral ischaemia is a complex disease process. Current therapeutic efforts are directed towards preventing tissue in with reversible ischaemia (the
ischaemic penumbra) progressing to full infarction. Without rapid treatment or spontaneous recanalisation, the penumbra will eventually infarct. There is currently debate regarding the imaging equivalent of the ischaemic penumbra and it has been suggested that combining diffusion-weighted and perfusion-weighted MR imaging might be helpful. The initial concept was based on the observation that abnormalities are often more extensive on perfusion imaging than on diffusion-weighted images. It was assumed that the abnormalities on diffusion-weighted images (DWI) were irreversible and represented the core of infarct. The ischaemic penumbra was thought to be the area of perfusion/diffusion mismatch surrounding the core. This concept has been challenged and recent reports showed that abnormalities on diffusion weighted images could be reversible. It appears likely that diffusion can already be impaired if the blood flow lies between 10–20 ml/100 g/min and not only below 10 ml/100 g/min, which is associated with irreversible ischaemic damage. This theory has been supported by animal experiments. There is a feeling that abnormalities on diffusion-weighted imaging should be interpreted with caution and may prove to be more frequently reversible with the more widespread use of thrombolysis. Others emphasise the need for better quantification of perfusion and diffusion parameters in order to differentiate reversible from irreversible ischaemia.

**Imaging of subacute and chronic stroke**

*Subacute stroke*

In the subacute phase, brain swelling may improve gradually but low attenuation change on CT and T₂ hyperintensities on MR persist. Gyriform contrast enhancement can be seen on CT and MR indicating disruption of the blood–brain barrier. Haemorrhagic transformation, defined as secondary bleeding into the ischaemic zone, is much more frequently detected on MRI than on CT. The occurrence and severity of haemorrhagic transformation correlates with the size of the infarct and degree of contrast-enhancement in the early stage and has been shown in up to 80% of infarcts on MRI.

A study using serial MR perfusion scans showed an increase of rCBV in the infarcted region in the subacute stage, often associated with recanalisation of the occluded vessel on MRA, which was followed by a decrease in rCBV in the chronic stage.

Abnormalities on diffusion-weighted imaging begin to normalise between 7–14 days; after this period, there is increased water mobility in gliotic tissue, which appears dark on diffusion-weighted images and bright on ADC maps.
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**Fig. 4** CT scans of a 75-year-old patient at the level of the lateral ventricles (a) and the vertex (b) showing evidence of small vessel ischaemic disease and two mature cortical infarcts, in the left and right frontal lobes. The T2*-weighted gradient-echo images at corresponding levels (c,d) show areas of low signal, consistent with haemosiderin staining, which indicates previous haemorrhage. The presence of multiple lobar haemorrhages is typical of amyloid angiopathy. Additional periventricular high signal areas are consistent with small vessel ischaemic disease.

Chronic stroke and transient ischaemic attack

The purpose of imaging in patients presenting with chronic stroke is not guidance of an acute therapeutic intervention, but secondary prevention of recurrent stroke.

In this context, it is important to establish the aetiology of the stroke. A distinction has to be made between previous haemorrhage, small vessel disease, large vessel disease and cardiac emboli. Establishing the diagnosis of an old haemorrhage is not possible with CT, since blood
clots become hypodense within a few weeks and may resemble infarcts. MRI is much more sensitive in showing evidence of previous haemorrhage because haemosiderin is taken up by macrophages and can persist for years, appearing as a characteristic low signal area on $T_2$-weighted images. The sensitivity for detection of haemosiderin can be increased by using $T_2^*$-weighted gradient-echo or echo planar imaging (EPI) sequences. Evidence of multiple peripheral haemorrhages in elderly patients is suggestive of amyloid angiopathy (Fig. 4). Small vessel ischaemic disease may be evident as hypodense white matter lesions on CT, but MRI is more sensitive to small vessel ischaemic changes, particularly if a FLAIR sequence is used. White matter lesions are more frequently seen in patients with hypertension, but may also be a feature of vasculitic diseases.

To determine the vascular territory of an infarct, one has to take into account variations of the circle of Willis, which can be readily assessed with MRA. One of the more frequently encountered variants is a fetal origin of the posterior cerebral artery from the internal carotid artery, which has been described in up to 31% of subjects. It is worth remembering that, in patients with this variant, occipital infarcts can be caused by carotid stenosis rather than vertebrobasilar disease.

Watershed infarcts at the boundaries between the anterior, middle and posterior cerebral artery territories (and between the deep and superficial cerebral circulation) may be the result of global ischaemia following an episode of hypotension or represent haemodynamic failure distal to a critically stenosed carotid artery.

Multiple infarcts in different vascular territories suggest cardiogenic emboli. Infarcts, which do not correspond to an arterial territory, have to raise the suspicion of venous infarcts, particularly if there is evidence of extensive haemorrhagic transformation. Patency of the major dural sinuses can be easily assessed with phase-contrast MR venography in these cases.

Assessment of the carotid bifurcation is crucial in the diagnostic work-up of chronic stroke and secondary prevention of a recurrent stroke. This can be performed non-invasively with Doppler ultrasound and MR angiography, which are discussed elsewhere in this issue.

Dissection of the carotid arteries is an important cause of stroke in young and middle aged adults. The characteristic MRI findings are a rim of high signal expanding the outer diameter of the artery and narrowing its lumen. The high signal represents subacute intramural haematoma, which becomes apparent after a few days. In the acute stage, the intramural haematoma is isointense to muscle and may be more difficult to detect.

MR perfusion imaging can also be useful in patients with chronic cerebrovascular disease and severe carotid artery stenosis. In cases with
established infarcts on T$_2$-weighted images, perfusion imaging can show more extensive abnormalities in regions that appear normal on T$_2$-weighted images, such as a prolongation of the MTT which may indicate tissue at risk. In the presence of bilateral carotid artery stenosis/occlusion, MR perfusion imaging may show impaired perfusion in a watershed distribution (see Fig. 1). In these cases, perfusion imaging can be used to monitor the results of an intra-extracranial bypass operation.

With significant carotid artery stenosis, cerebral perfusion imaging may be normal at rest, but show impairment following a challenge of the vascular reserve with acetazolamide. Normally, the cerebral flow increases following acetazolamide administration. Some patients with severe steno-occlusive disease have a decreased response to the acetazolamide challenge$^{44}$. Demonstration of this unilateral vascular steal on xenon CT perfusion studies has been shown to be associated with a 30% stroke rate during follow-up$^{45}$.

Finally, one can assess recovery from stroke with functional MRI using cortical activation studies. This can provide evidence of the plasticity of the brain, e.g. when the function of areas damaged by ischaemia is taken over by adjacent or contralateral cortical regions$^{46}$.

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