

## Supplementary material

### Imaging reading methods

The readings described below were performed as consensus readings of two readers (one radiology resident and one neurology stroke fellow). Images were reviewed under appropriate conditions (dimmed light, multi monitor setup, windowing tools). Between the non-contrast head computed tomography (NCCT), the multi-phase computed tomography angiography (CTA) and the follow-up readings, reading was paused for 2 weeks respectively.

#### *NCCT reading*

5 mm reconstructed axial unenhanced head computed tomography (CT) images were available with sagittal and coronal reformations. The readers had access to the side that was clinically affected, the National Institutes of Health Stroke Scale (NIHSS) score and the time of symptom onset and time of CT acquisition. The readers were blinded to the CTA, computed tomography perfusion (CTP), digital subtraction angiography (DSA), follow-up magnetic resonance (MR) or CT images and to clinical outcome data.

#### Recorded items

- 1) Alberta Stroke Program Early CT Score (ASPECTS): The ASPECTS was scored on NCCT images to assess the extent of early ischemic changes. Areas with early ischemic changes (relative hypodensity, loss of grey white matter differentiation, and effacement of sulci) were considered as affected and the regions affected were reported explicitly.
- 2) Affected side: The affected side (side of early ischemic changes and/or side of hyperdense or unsure hyperdense vessel sign) was reported. In case of early ischemic changes in the anterior circulation it was reported whether the right or left side was affected. In case of supratentorial and cerebellar posterior circulation changes, the affected side (right or left) was reported as well. In case of brainstem early ischemic changes, no side was reported.
- 3) Baseline hemorrhage: The presence of hemorrhage (defined as hyperdense foci with a typical morphology and blood equivalent Hounsfield unit [HU] values of 50–80) on the baseline NCCT was reported and in case of a (1) parenchymal hemorrhage, the side (right, left), location (frontal, parietal, temporal, occipital, limbic lobe, insula, deep grey matter, cerebellum, pons, mesencephalon, medulla); (2) subarachnoid hemorrhage, the side (right, left), location (frontal, parietal, temporal, occipital, limbic lobe, insula, infratentorial); (3) subdural hemorrhage, the side (right, left), location

(frontal, parietal, temporal, occipital, infratentorial); (4) epidural hemorrhage, the side (right, left), location (frontal, parietal, temporal, occipital, infratentorial); and (5) intraventricular hemorrhage, the side (right, left, midline), affected ventricles/cisterns (right or left lateral ventricle, 3rd ventricle, aqueduct, 4th ventricle, prepontine/perimesencephalic or any other cistern) was reported.

- 4) Subacute infarcts: The presence of subacute infarcts (defined as sharply hypodense demarcated areas with a typical vascular distribution) was recorded. In case subacute infarcts were present, the side (right or left) and ASPECTS region in which the subacute infarct was present was recorded. In case of subacute infarcts in the posterior circulation, for supratentorial and cerebellar lesions, the side (right or left) and territory (occipital lobe, mesiotemporal lobe, deep grey matter, cerebellum) was reported. For brainstem lesions, no side was reported.

- 5) Hyperdense vessel sign: The presence of a hyperdense vessel sign was reported. It was noted whether a clear, unambiguous hyperdense vessel or no hyperdense vessel was seen.

Ambiguous cases were reported as unsure. In case of presence of a hyperdense or unsure hyperdense vessel sign, the vessel was noted as follows: (1) internal carotid artery (ICA; intracranial ICA); (2) middle cerebral artery (MCA) (hyperdensity which extends into the M1 and M2 portion); (3) M1 (hyperdensity only in the presumed localization of the M1 portion); (4) M2 (hyperdensity only in the presumed localization of the M2 portion); (5) B (hyperdensity only in the presumed localization of the basilar artery); (6) P1 (hyperdensity only in the presumed localization of the P1 portion); (7) P2 (hyperdensity only in the presumed localization of the P2 portion); and (8) V (hyperdensity only in the presumed localization of the vertebral artery).

#### *Multiphase CTA reading*

Five millimeter reconstructed axial unenhanced head CT images were available with sagittal and coronal reformations. Multiphase CTA images. Multiphase images (section thickness 0.625 mm) were also available; an arch to vertex CTA constituted the first phase that was acquired during the peak arterial phase in the healthy brain parenchyma and was triggered by bolus monitoring. Coronal and sagittal reformations for the first phase were available. The second (peak venous) and third (late venous) phase were acquired after 11 and 19 seconds. Eighty milliliter of contrast were injected at a rate of 5 mL/sec followed by a 50 mL saline bolus at a rate of 6 mL/sec. Axial images were reconstructed for all three phases with 1 mm overlap and multiplanar reconstructions for axial, coronal, and

sagittal images of the circle of Willis were performed with 3-mm thickness at 1-mm intervals. Thick-section axial maximum intensity projections at 24-mm thickness and 4-mm intervals were also reconstructed and reviewed.

#### Recorded items

- 1) Affected side: The side (right or left) of the vessel occlusion was reported in case of anterior circulation occlusions and posterior cerebral artery (PCA) occlusions. In case of vertebral and basilar occlusions, no side provided. In case of no evident vessel occlusion, a side was reported if there were clear early or subacute ischemic changes on one side in the infra- or supratentorial parenchyma. In case of venous thrombosis, the side was provided for the sigmoid and transverse sinus, whereas no side was provided for the superior, inferior sagittal sinus and sinus rectus.
- 2) Primary occlusion: The primary occlusion was defined as the most proximal occlusion that subsequently affects the largest brain parenchyma volume. The most proximal end of the clot was decisive for the clot localization (e.g., a clot that extends from the ICA in the M1 and M2 was reported as an ICA occlusion). The vessels were noted as follows: (1) ICA: Intra- or extracranial ICA. The beginning was defined as the carotid bifurcation and the end as the ICA bifurcation; (2) M1: Begins distal to the ICA bifurcation and ends at the main bifurcation in the inferior and superior M2 branch/at the trifurcation in case of a trifurcation; (3) Proximal M2: M2 segment extending from the MCA main bifurcation up to 1 cm distal to the end of the M1 segment, easily accessible for endovascular treatment; (4) Distal M2: M2 segment more distal than 1 cm distal to the end of the M1 segment, ends at the circular sulcus of the insula; (5) M3: Begins at the circular sulcus of the insula and ends at the external surface of the Sylvian fissure; (6) M4: Begins distal to the M3 that reaches the external surface of the Sylvian fissure, superficially coursing branches at the lateral convexity; (7) A1: Starts at the ICA bifurcation, ends at the origin of the anterior communicating artery; (8) A2: Starts at the origin of the anterior communicating artery, ends at the genu of the corpus callosum; (9) A3: Starts at the genu of the corpus callosum, ends at the posterior turn of the artery, where it courses above the corpus callosum; (10) A4: Branches above the body of the corpus callosum; (11) P1: Origins at the terminal bifurcation of the basilar artery, ends at the origin of the posterior communicating artery; (12) P2: Starts at the origin of the posterior communicating artery and courses around the mesencephalon ends when the vessel enters the quadrigeminal cistern; (13) P3: Starts when the vessel enters

the quadrigeminal cistern, courses in the quadrigeminal cistern; (14) P4: Starts when the branches enter the cortex, cortical branches; (15) Basilar artery: Starts at the junction of the vertebral arteries and ends at the basilar bifurcation; and (16) Vertebral artery: Start at its origin from the subclavian artery (or aortic arch on the left side) and ends at the vertebrobasilar junction.

- 3) Clot burden score: The clot burden score was reported in patients with anterior circulation vessel occlusion. In case of no evident occlusion, the clot burden score was not reported. In case posterior circulation and distal anterior circulation (M3 and M4, A2, A3, A4) occlusions, the clot burden score was reported as 10.
- 4) Secondary occlusion: In case of a second, major occlusion with clear flow affection in the downstream territory attributable to this occlusion, this was noted as a secondary occlusion. The occlusion localization was noted as for primary occlusions.
- 5) Side of secondary occlusion: The occlusion side was noted as for primary occlusions (right or left for anterior circulation and PCA occlusions, no side was reported for vertebral and basilar occlusions).
- 6) Pial arterial filling (collateral grading): The delay and extent of collateral filling for the primary occlusion was graded on axial maximum intensity projections of the three multi-phase computed tomography angiography phases. Grading was as follows: (Grade 0) Poor collaterals: no or only few vessels visible in any phase within the occluded vascular territory compared to the asymptomatic contralateral hemisphere; (Grade 1) Intermediate collaterals: delay of two phases in filling in of peripheral vessels with or without decreased prominence and extent or a one-phase delay and some ischemic regions with no only few or no vessels compared to the asymptomatic contralateral hemisphere; (Grade 2) Good collaterals: no delay or 1 phase delay in filling of peripheral vessels with identical or increased prominence of vessels compared to the asymptomatic contralateral hemisphere.

In case of posterior circulation occlusions, this item was not reported.

#### Follow-up reading

Either magnetic resonance imaging (MRI) (susceptibility-weighted or gradient echo images, diffusion weighted images and apparent diffusion coefficient images and fluid attenuated inversion recovery images) or unenhanced head CT images that were obtained 24 ( $\pm 6$ ) hours after initial imaging were reviewed. The readers had access to the side that was clinically affected, but were blinded to baseline NCCT; CTA and CTP im-

ages and DSA images.

#### Recorded items

- 1) Imaging modality: It was noted whether the follow-up imaging was either a CT or an MR. In case both existed, the MR was chosen for evaluation.
- 2) Final ASPECTS: The follow-up ASPECTS was scored. A region was called affected when at least 30% of it was affected and the affected regions were reported explicitly. (1) CT: A region was called affected when there was clear, sharp hypodense demarcation of at least 30% of the parenchyma, with or without hemorrhage. Regions with changes indicative of older infarcts like encephalomalacia or old calcified infarcts were not considered affected. (2) MRI: A region was called affected if there was DWI-hyperintense signal in at least 30% of the parenchyma. In case of bilateral lesions, the side with the larger affected territory was reported. In case of ischemic changes in the posterior circulation and in the anterior cerebral artery (ACA) territory, the ASPECTS was recorded as 10.
- 3) Side: The side of ischemic changes were reported for anterior circulation and PCA territory infarcts. For brainstem lesions, no side was reported. In case of bilateral ischemic changes, the side with the larger affected territory was reported. If the extent of ischemic changes was equal on both sides, this was reported as both sides. In case no new or subacute ischemic lesions could be identified, this was reported as no changes.
- 4) Ischemic changes in the ACA territories: It was reported whether ischemic changes in the ACA territory were present (either isolated or in addition to ischemic changes in other territories). In case they were present, the side (left, right, or both) was reported.
- 5) Ischemic changes in the vertebrobasilar territory: It was reported whether ischemic changes in the vertebrobasilar territory were present (either isolated or in addition to ischemic changes in other territories). In case they were present, for PCA territory lesions, the side (left, right, or both) was reported. For brainstem lesions, no side was reported.
- 6) Hemorrhagic transformation: For CT, the presence and degree of hemorrhagic transformation according to the The European Cooperative Acute Stroke Study (ECASS) criteria and an extension of the classification was used for reviews

of follow-up MRIs. The scans were assigned to each of the following categories: no hemorrhagic transformation, hemorrhagic infarction type 1, hemorrhagic infarction type 2, parenchymal hemorrhage type 1, parenchymal hemorrhage type 2. In case both MR and CT follow-up imaging was available, MRI was used. In order to distinguish contrast staining post thrombectomy and hemorrhage on CT, density measurements of the affected area were taken and areas with HU values 50–80 considered as hemorrhage, whereas much higher values (above 120 HU) were considered contrast staining.

- 7) Remote parenchymal hemorrhage: The presence of parenchymal hemorrhage in areas remote from the ischemic changes was reported. In case they were present, the side (right, left) and location (frontal, parietal, temporal, occipital, limbic lobe, insula, deep grey matter, cerebellum, pons, mesencephalon, medulla) was reported.
- 8) Other hemorrhage: The presence and type of extraaxial hemorrhage (subarachnoid, subdural, epidural, intraventricular) was reported. In case an extraaxial hemorrhage was present, the side (right, left) and location (frontal, parietal, temporal, occipital, limbic lobe, insula, infratentorial) was reported.

#### Additional statistical methods (reclassification analysis)

The net reclassification index (NRI) and integrated discrimination improvement (IDI) were calculated to compare imaging paradigms in predicting outcome to provide an estimate of the relative incremental benefit of each imaging approach. The NRI assigns a positive value for a model that correctly reclassifies a patient, while those who are incorrectly reclassified are assigned a negative value. Thus, a positive NRI indicates reclassification improvement compared to the base model, while a negative value indicates worsening. The magnitude of the NRI corresponds to the extent of improvement/worsening. The IDI represents the difference in discrimination slopes: it expresses how much a new model increases the assigned risk in patients with events and decreases the assigned risk in patients without events compared to the base model. Reclassification analysis was attempted for both outcomes.

Two-sided *P*-values <0.05 were considered statistically significant.