7. COMPLICATIONS AFTER CAROTID INTERVENTIONS

7.1. Peri-operative

7.1.1. Stroke after carotid endarterectomy

7.1.1.1. Intra-operative. Intra-operative stroke is a new neurological deficit (worsening of pre-existing deficit), apparent following recovery from anaesthesia (or during CEA under LRA), lasting > 24 hours. Most follow intra-operative embolisation (carotid mobilisation, shunt insertion, flow restoration, accumulation of thrombus on endarterectomy zone). A minority (20%) are haemodynamic after carotid clamping or shunt malfunction.[499] In a 21 year audit (n = 2 300), most intra-operative strokes followed embolisation of luminal thrombus at flow restoration, with the source being bleeding from transected vasa vasorum onto the endarterectomised surface.[309] One advantage of CEA under LRA is that the timing of new deficits can be accurately determined. For patients undergoing CEA under GA, abrupt EEG changes predict the likeliest time of onset.[500] Patients with a triad of hemiplegia, homonymous hemianopia, and higher cortical dysfunction on recovery from anaesthesia are likely to have suffered ICA or MCA occlusion. If one to two triad components are present, occlusion of one or more MCA branches is likely.[501]

Previously, patients recovering from anaesthesia with a new neurological deficit underwent immediate re-exploration to exclude thrombus within the endarterectomy zone. This remains the recommendation in the 2021 SVS guidelines.[4] However, a recent Delphi consensus study concluded that immediate re-exploration remained appropriate in patients experiencing a new deficit when flow was restored with CEA under LRA, but in all other peri-operative phases, rapid imaging of carotid vessels and brain was advised before re-exploration.[502] In ACST-1, there was no difference in rates of disabling/fatal stroke between patients who underwent immediate re-exploration *versus* those who did not.[503] The priority, therefore, is to quickly identify patients with ICA thrombosis, as they will benefit from immediate re-exploration. TCD aids decision making, as MCA velocities with ICA thrombosis are identical to those during carotid clamping. Thrombosis is also preceded by increasing rates of embolisation.[309] DUS can confirm flow in the endarterectomy zone, but subcutaneous air makes it difficult to interpret early post-operative findings. At re-exploration, thrombus should be removed. If thrombus extends distally, it should be carefully removed with a Fogarty catheter. Following thrombectomy, technical errors are corrected, and a completion angiogram performed. Embolic occlusion of the ipsilateral anterior or middle cerebral artery can be treated by re-exploration (to remove thrombus in the endarterectomy zone) followed by intra-arterial thrombolysis.[504] Emergency MT is another option in patients with embolic MCA mainstem occlusion. No RCTs have been done, but targeted intra-operative neuromonitoring (TCD, EEG) and QC assessment (completion angioscopy, DUS, angiography) have been associated with significant reductions in intra-operative stroke.[71,309,500,505]

7.1.1.2. Post-operative. This is defined as a new neurological deficit (or worsening of a pre-existing deficit) after an uneventful recovery from anaesthesia, with symptoms lasting > 24 hours. In the first six hours, the most common cause is ICA thrombosis or embolism from mural thrombus in the endarterectomy zone. A Delphi consensus recommended rapid imaging before re-exploration.[502] After six hours, CT and extracranial and intracranial CT/CTA will exclude ICA thrombus, cerebral oedema, or parenchymal haemorrhage. In ICSS, the commonest cause of post-operative stroke was hyperperfusion syndrome (HS).[385] HS is discussed in more detail in section 7.1.3.5.

7.1.1.3. Predictors of stroke after carotid endarterectomy. In ECST, predictors included (i) female sex (10.4% *vs.* 5.8%, p = .001); (ii) PAD (12.0% *vs.* 6.1%, p = .001); (iii) pre-operative SBP (< 120 mmHg = 3.4%; 121 − 159 = 6.5%; 160 − 180 = 7.7%; > 180 mmHg = 13.0%, p = .040); and (iv) presentation (retinal [3.2%], hemispheric stroke [6.3%], TIA [9.1%], p = .006).[352] Predictive features in NASCET were (i) hemispheric *versus* retinal events (6.3% *vs.* 2.7%; OR 2.3; 95% CI 1.1 − 5.0); (ii) left *versus* right CEA (6.7% *vs.* 3.0%; OR 2.3, 95% CI 1.4 − 3.6); (iii) contralateral occlusion (9.4% *vs.* 4.4%; OR 2.2, 95% CI 1.1 − 4.5); (iv) ipsilateral CT/MR infarct (6.3% *vs.* 3.5%; OR 1.8; 95% CI 1.2 − 2.8); and (v) irregular *versus* smooth plaques (5.5% *vs.* 3.7%; OR 1.5, 95% CI 1.1 − 2.3).[506] In ICSS, stroke was more frequent in females (RR 1.98; 95% CI 1.02 − 3.87, p = .05) and with increased DBP (RR 1.30 per +10 mmHg; 95% CI 1.02 − 1.66, p = .04), but unrelated to CEA method or GA *versus* LRA.[507] In a multivariable model, increased DBP was the only independent predictor of stroke, MI, or death.[507] In ACST-1, DBP was also an independent predictor for stroke.[13]

Recommendation 91			New
For patients experiencing a peri-operative stroke, it is recommended to differentiate between an intra-operative and a post-operative stroke.			
Class	**Level**	**References**	**ToE**
I	C	Meershoek *et al.* (2021)[502]	

Recommendation 92			New
For patients who develop an ipsilateral neurological deficit after flow is restored following carotid clamp release when carotid endarterectomy is performed under locoregional anaesthesia, immediate re-exploration of the carotid artery is recommended.			
Class	**Level**	**References**	**ToE**
I	C	Meershoek *et al.* (2021)[502]	

7.1.2. Stroke after carotid artery stenting.

In a meta-analysis of SCS patients in RCTs, the risk of stroke on the day of CAS was 4.7% with an additional 2.5% during days 1 − 30. Most were ischaemic (94%), with 91% ipsilateral to the stented ICA.[48] Important causes include embolisation, in stent thrombosis, ICA/CCA dissection, HS, and ICH.