Recommendation 20			Unchanged
For average surgical risk patients with an asymptomatic 60–99% stenosis in the presence of one or more imaging or clinical characteristics that may be associated with an increased risk of late stroke*, carotid stenting may be an alternative to carotid endarterectomy, provided 30 day stroke/death rates are ≤3% and patient life expectancy exceeds five years.			
Class	**Level**	**References**	**ToE**
IIb	B	Mannheim *et al.* (2017)[222], Rosenfield *et al.* (2016)[224], Eckstein *et al.* (2016)[225], Nicolaides *et al.* (2005)[261], Kakkos *et al.* (2013)[264], Kakkos *et al.* (2009)[270], Kakkos *et al.* (2014)[271], Hirt *et al.* (2014)[272], Nicolaides *et al.* (2010)[273], Gupta *et al.* (2013)[274], King *et al.* (2011)[275], Gupta *et al.* (2015)[276], Markus *et al.* (2010)[277], Topakian *et al.* (2011)[278], Silver *et al.* (2011)[280]	

* See Table 8 for imaging/clinical criteria conferring an increased risk of stroke on BMT in ACS patients.

Recommendation 21			Unchanged
For asymptomatic patients deemed by the multidisciplinary team to be 'high risk for surgery' and who have an asymptomatic 60–99% stenosis in the presence of one or more imaging/clinical characteristics that may be associated with an increased risk of late stroke on best medical therapy, carotid stenting may be considered provided anatomy is favourable, 30 day death/stroke rates are ≤3% and patient life expectancy exceeds five years*.			
Class	**Level**	**References**	**ToE**
IIb	B	Gurm *et al.* (2008)[223], Nicolaides *et al.* (2005)[261], Kakkos *et al.* (2013)[264], Kakkos *et al.* (2009)[270], Kakkos *et al.* (2014)[271], Hirt *et al.* (2014)[272], Nicolaides *et al.* (2010)[273], Gupta *et al.* (2013)[274], King *et al.* (2011)[275], Gupta *et al.* (2015)[276], Markus *et al.* (2010)[277], Topakian *et al.* (2011)[278], Yadav *et al.* (2004)[282]	

* See Table 8 for imaging/clinical criteria conferring an increased risk of stroke on BMT in ACS patients.

3.10. Carotid revascularisation and cognitive impairment

Five per cent of patients aged > 60 have dementia. Globally, the annual cost of treating dementia exceeds $US 1 trillion (€ 816 billion) and may reach $US 2 trillion (€ 1.6 trillion) by 2030.[287] In 20% of dementia patients, atherosclerosis or other occlusive diseases affecting cerebral vessels is responsible (vascular dementia), while 20–30% have vascular dementia and Alzheimer's.

3.10.1. Do asymptomatic carotid stenoses cause cognitive impairment? There is speculation that ACS may be responsible for cognitive decline. In a 2013 systematic review, nine out of 10 observational studies reported an association between ACS and cognitive impairment,[50] but there was no further scrutiny as to whether this translated into a causal association. In a larger systematic review (35 observational studies; 3 626 ACS patients, 10 936 controls), 33/35 studies (94%) reported an association between ACS and cognitive impairment.[87] However, such association does not necessarily mean ACS has an aetiological role *versus* being a marker for something else. The systematic review examined the evidence and was unable to unequivocally demonstrate that ACS was causally associated with cognitive dysfunction via involvement in the pathophysiology of white matter hyperintensities on MRI, lacunar infarction or via an embolic mechanism.[87] Surprisingly few studies have evaluated the relationship between ACS, ipsilateral cortical infarction, and cognitive impairment. An alternative mechanism whereby ACS might cause cognitive impairment is haemodynamic. As the ACS becomes more severe, patients with a non-functioning CoW and poor collateralisation compensate by vasodilation of ipsilateral intracranial arterioles. This maintains cerebral blood flow, but a point arises where arterioles cannot dilate further. The patient then enters a state of impaired then exhausted cerebral vascular reserve (CVR) with limited (or no) capacity to vasodilate further and blood flow then starts to decline. CVR can be measured using single photon emission tomography, positron emission tomography, or TCD monitoring of ipsilateral mean middle cerebral artery (MCA) velocities during CO_2 inhalation or breath holding (which raises blood CO_2 levels), which causes vasodilatation and increased MCA velocities, but only if CVR is not exhausted.

Ten studies have evaluated the relationship between impaired CVR and cognitive impairment, with 90% reporting at least one test of impaired cognition.[87] There was a stepwise increase in severity of cognitive impairment from normal in patients with severe ACS plus normal CVR (bilaterally), through unilateral impaired CVR (increased cognitive impairment), with maximum cognitive dysfunction in patients with bilateral impaired CVR.[288] Patients with severe ACS (unilateral or bilateral) and normal CVR had cognitive scores similar to controls.[289,290] Finally, patients with severe ACS and impaired CVR were more likely to suffer further cognitive decline over time *versus* patients with severe ACS and normal CVR.[288,291–293]

3.10.2. Do carotid interventions improve cognition function? A second systematic review (31 observational studies) evaluated the effect of carotid interventions on early and late post-operative cognition in ACS patients.[46] Assessment of early cognitive function was defined as re-assessment within three months after CEA or CAS (*vs.* baseline). Assessment of late cognitive function involved assessment at least five months after CEA or CAS. In 13/21 cohorts, late reassessment was at least one year after baseline.[46] Table 12 details the effect of carotid interventions on early post-operative cognition in 24 patient cohorts (11 CEA; 10 CAS; 3 CEA + CAS), and late cognitive function in 21 patient cohorts (12 CEA; 7 CAS; 2 CEA + CAS).[46]