persist, at which time a modified Rankin scale score should be repeated to quantitate the degree of recovery.

Other major adverse cardiovascular events occurring within 30 days of dissection onset in the case of medical management or 30 days of surgical intervention should likewise be reported, as should a composite of major adverse cardiovascular events, which includes myocardial infarction, stroke, and death.

Imaging for stroke. In cases in which imaging is obtained, evidence of new cerebral infarct or hemorrhage on CT or MRI, even in the absence of discernible neurologic deficit, also indicates the presence of acute stroke.[65,72] Efforts should be made to separate ischemic from hemorrhagic strokes as subsequent treatment schemes will differ.[73,74] Ischemic strokes are due to infarction of central nervous system tissue, whereas hemorrhagic infarcts are characterized by intraparenchymal, intraventricular, or subarachnoid hemorrhage. Further characterization of strokes radiographically can lend insight into the mechanism of stroke. This holds importance in attributing stroke to an embolic or watershed etiology. The delineation of anterior vs posterior strokes as well as unilateral vs bilateral also lends insight into the pathophysiologic mechanism of stroke as the vascular distribution implicates different vessels (carotid vs vertebral artery) and causes. For example, occurrence of a unilateral posterior circulation stroke after left subclavian coverage without revascularization during TEVAR may indicate posterior circulation hypoperfusion. However, bilateral posterior circulation stroke in the same setting may indicate an embolic cause related to arch manipulation and not affected by the presence or absence of subclavian revascularization.[75] Stroke distribution has also been shown to have an impact on mortality. Previous studies have shown that posterior strokes have higher rates of morbidity and early mortality than anterior strokes.[76] As noted before, laterality should be noted as well, and as branched devices are being developed, studied, and used, these anatomic details will become increasingly important.

After the diagnosis of stroke, any additional imaging performed to rule out potential causes of stroke, such as imaging of the carotid arteries or echocardiography to rule out a cardiac source (eg, intracardiac thrombus or patent foramen ovale with or without atrial septal aneurysm), should be documented.

Acute kidney injury. The definition and classification scheme for acute kidney injury should encompass any decline in kidney function as well as the need for dialysis, whether transient or permanent. As noted before (Section 4, Renal Malperfusion), the etiology of renal dysfunction in patients with aortic dissection is multifactorial and can include renal malperfusion, contrast nephropathy, the sequelae of medical anti-impulse therapy in patients with chronic hypertension, and possible embolization. The aforementioned AKIN grading scheme,[77] a consensus classification developed by intersocietal collaborations between nephrology and critical care, consists of three stages that take into account changes in serum creatinine concentration as well as urine output: stage 1, increase in serum creatinine concentration 1.5- to 2-fold; stage 2, increase in serum creatinine concentration 2- to 3-fold; and stage 3, serum creatinine concentration increase >3-fold or anuria for ≥12 hours (Table IV). In instances in which serum creatinine concentration is unknown at baseline, urine output should be the primary determinant of grade using AKIN. In addition, it should be noted whether there is a need for dialysis and whether this need is temporary in-house only, present at discharge, or permanent on follow-up.

SCI. Multiple causes of SCI have been identified after aortic dissection (see Section 4, Stroke and Spinal Cord Ischemia), including sequelae of the dissection itself and its treatment. Because of the variability of the collateral network in the thoracolumbar spinal cord, endovascular coverage or surgical sacrifice of intercostal or lumbar arteries can cause SCI by compromised flow in watershed areas. Because SCI has also been noted in individuals with patent radicular arteries, the importance of perfusion pressure in the development of SCI should be noted as well.

Spinal cord perfusion pressure (SCPP) is defined by the equation SCPP = MAP − CSF pressure (or central venous pressure, whichever is higher). SCPP in patients suffering SCI should be documented, if feasible, as well as any attempts to manipulate SCPP by raising the MAP or lowering the CSF pressure or central venous pressure. Another cause of SCI is embolization to segmental arteries supplying the spinal cord. Patients with SCI can have varying presentations as described in Section 4, with variable severity, onset, and potential for recovery. Time at onset should be noted as this could implicate a dissection-related vs early perioperative (extent of endovascular coverage, surgical ligation, or embolization) vs delayed perioperative cause, where hemodynamic instability in a patient initially neurologically intact after surgical repair could be the cause.

The modified Tarlov scoring system as discussed in Section 4 (Table VI) is a useful grading scheme for SCI and includes scores of 0 to 5, which encompass the full spectrum of SCI.[78,79] Any degree of SCI related to either the index dissection or its management should be documented using this system. It is also important to note whether there is improvement in functional status at the time of discharge or in late follow-up. Patients with SCI with subsequent improvement at discharge have been shown to have lasting neurologic recovery.[80] Consequently, a Tarlov score should also be documented for all patients with SCI at the time of discharge and in late follow-up.