Perioperative Care of Patients at High Risk for Stroke during or after Non-Cardiac, Non-Neurologic Surgery: Consensus Statement from the Society for Neuroscience in Anesthesiology and Critical Care

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Abstract: Perioperative stroke can be a catastrophic outcome for surgical patients and is associated with increased morbidity and mortality. This consensus statement from the Society for Neuroscience in Anesthesiology and Critical Care provides evidence-based recommendations and opinions regarding the preoperative, intraoperative, and postoperative care of patients at high risk for the complication.

Key Words: stroke, ischemic stroke, perioperative stroke, cerebrovascular, cerebrovascular accident, noncardiac surgery, neurologic complication

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Stroke can be a catastrophic outcome for patients undergoing noncardiac, nonneurologic surgery and is associated with an adjusted 8-fold increase in mortality.[1] Unlike stroke in the community setting, the mechanistic cascade leading to perioperative stroke has a discrete and highly-predictable origin: surgical intervention. Given the fact that surgery and anesthesia are associated with an increased risk of stroke compared to nonsurgical controls,[2] establishing perioperative recommendations to minimize risk could be impactful. Stroke after noncardiac, nonneurologic surgery is relatively understudied and there is a need for clarifying the clinical management of surgical patients at high risk for the complication.

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METHODOLOGY

Definition of Perioperative Stroke

Sacco et al have developed a consensus statement regarding the broad definition of stroke.[3] However, for the purposes of this consensus statement, "perioperative stroke" is defined as a brain infarction of ischemic or hemorrhagic etiology that occurs during surgery or within 30 days after surgery. We recommend that such a standardized definition be adopted for future reports. It is important to note that this clinical situation is distinct from that of a patient presenting for acute therapy after a stroke has occurred in a nonoperative setting. Periprocedural care of patients presenting for endovascular interventions to treat stroke is described elsewhere.[4]

Purpose of the Consensus Statement

The purpose of this consensus statement is to provide evidence-based recommendations regarding (1) preoperative identification of patients at high risk of stroke during or after noncardiac, noncarotid, nonneurologic surgery, (2) preoperative considerations to mitigate risk, (3) intraoperative management to mitigate risk, and (4) appropriate steps for clinical care if stroke is identified in the postoperative period.

Focus

Patients undergoing carotid endarterectomy and a variety of cardiac surgeries are known to be at high risk for perioperative stroke, with fairly clear etiologies (e.g., embolic event).[5] As such, there has been considerable attention to the prevention of stroke in these populations. The focus of the current consensus statement is the prevention and management of ischemic stroke in adult patients undergoing noncardiac, noncarotid, and nonneurologic surgery.

Application

This consensus statement is intended for use by anesthesiologists, anesthesia providers, surgeons and other perioperative care providers. It may also serve health care professionals such as internists or neurologists who evaluate

patients in the perioperative period. For the purposes of this article, anesthesia care refers to general anesthesia, regional anesthesia, or monitored anesthesia care.

Task Force Members and Consultants

The initial recommendations were developed by five anesthesiologists with expertise in clinical neuroscience and neuroanesthesiology, practicing in academic departments across the U.S. These individuals were chosen from membership of the Society for Neuroscience in Anesthesiology and Critical Care (SNACC), an international organization. Applicants were required to have published peer-reviewed research on the subject of perioperative stroke or have documented experience in the care of patients with stroke. An outline of the proposed consensus statement was developed and approved by the Executive Committee and Board of Directors of SNACC. The task force members agreed on criteria for evidence and then evaluated peer-reviewed studies pertaining to perioperative stroke. Recommendations were developed by the task force and then assessed by the Executive Committee of SNACC. After incorporating input from the Executive Committee, the Task Force presented the draft guidelines to the international membership of SNACC through its website; thirty days were allowed for suggested revisions. This input and all further available information were incorporated into the consensus statement. The consensus statement was then reviewed and, after further revision, was supported by the American Society of Anesthesiologists.

AVAILABILITY AND STRENGTH OF EVIDENCE

This consensus statement was informed by published literature obtained through PubMed and other health-care databases, direct internet searches, task force members, and manual searches of references found in review articles. Peer-reviewed articles were considered that provided either scientific evidence (e.g., randomized controlled trial, RCT) or opinion-based evidence.

a) Scientific Evidence

(i) Category A: Supportive Literature
1. Level 1: The literature contains multiple RCTs and findings are supported by meta-analysis.
2. Level 2: The literature contains multiple RCTs, but no meta-analysis is possible.
3. Level 3: Only one RCT exists in the literature.

(ii) Category B: Suggestive Literature
1. Level 1: The literature contains observational comparisons of interventions (e.g., case-control study) that indicate statistically significant differences with respect to the outcome of perioperative stroke.
2. Level 2: The literature contains noncomparative observational studies with associative or descriptive statistics.
3. Level 3: The literature contains case reports.

(iii) Category C: Equivocal Literature
1. Level 1: Meta-analysis did not find significant differences.
2. Level 2: No meta-analysis is possible; RCTs found inconsistent evidence.
3. Level 3: Observational studies report inconsistent findings that do not permit inference.

(iv) Category D: Insufficient Evidence from the Literature
1. Silent: No identified studies exist on the relationship between intervention and perioperative stroke.
2. Inadequate: The literature does not permit clear interpretation of findings due to methodological considerations.

Opinion-based Evidence

Category A: Expert opinion from task-force consultants.
Category B: Membership opinion obtained from survey.
Category C: Informal opinion from open-forum testimony, internet-based comments, and other communications.

PREOPERATIVE RECOMMENDATIONS

Identifying patients at high risk of stroke

In order to prevent perioperative stroke, it is critical to identify those at high risk for the complication. The incidence of stroke in a broad surgical population (excluding cardiac, carotid, major vascular and neurologic surgery) is approximately 1 per 1000 cases[1] and approximately 6 per 1000 cases after major vascular surgery below the diaphragm[6]; perioperative stroke increased length of hospital stay and risk of death. These data are derived from more than 550,000 patients across two studies of the American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP) database, which is currently the highest quality dataset available for the epidemiology of perioperative stroke incidence and outcome. The incidence of perioperative stroke stratified by case type can be found in Table 1. The significant majority of perioperative strokes are ischemic rather than hemorrhagic[7,8]; the ACS-NSQIP database does not distinguish between the two. Preliminary data of the NeuroVision trial, conducted in noncardiac surgery patients with cardiovascular risk factors, suggest that the incidence of *covert* stroke (i.e., without obvious deficit) is 10%, as identified by magnetic resonance imaging in the postoperative period.[9] If confirmed by the larger trial, this finding could have important implications for the study and prevention of perioperative stroke after noncardiac surgery.

Due to the relative rarity of overt perioperative stroke in the noncardiac population, prospective identification of risk factors has been limited. Data regarding risk profile have therefore been derived from case series, case-control studies, or large database investigations. Table 2 shows the independent predictors of perioperative stroke found in recent major studies of the complication. Three of the most consistent risk factors for perioperative stroke identified in the literature are advanced age, renal failure, and a history of stroke or transient ischemic attack.[1,6,10–14] In general terms, all patients presenting for surgery with a history of cerebrovascular compromise should be regarded as high risk for perioperative stroke.

TABLE 1. Incidence of Stroke for Noncardiac, Nonvascular, Nonneurologic Surgeries

	Stroke – All Age %, (n)	Stroke – Age ≥ 65 %, (n)
Bateman, et al, 2009; Nationwide Inpatient Sample		
Hip Arthroplasty (N = 1,568)	0.4 (6)	0.5 (5)
Lung Resection (N = 1,484)	0.3 (5)	0.7 (5)
Colectomy (N = 33,426)	0.4 (130)	0.7 (100)
Mashour et al, 2011; American College of Surgeons- National Surgical Quality Improvement Program		
Hepatobiliary – Biliary Tree (N = 43,289)	0.1 (36)	0.2 (23)
Excisional breast (N = 36,793)	0.0 (16)	0.1 (11)
Hernia – Ventral/Umbilical/Incisional/Other (N = 32,638)	0.1 (28)	0.3 (21)
Hernia – Inguinal/Femoral Incisional Mesh (N = 26,448)	0.1 (17)	0.1 (10)
Colorectal – Appendectomy (N = 26,046)	0.0 (6)	0.2 (4)
Esophagogastric – Bariatric (N = 23,766)	0.0 (5)	0.0 (0)
Head and Neck – Tumor (N = 20,057)	0.0 (7)	0.1 (3)
Minor Vascular - Chest/Extremity (N = 5,883)	0.0 (2)	0.1 (1)
Small Intestine – Resection/Ostomy (N = 5,860)	0.5 (27)	0.6 (14)
Small Intestine – Lysis of adhesions, other (N = 5,683)	0.3 (17)	0.7 (14)
Abdominal – Exploration (N = 5,760)	0.5 (26)	0.9 (18)
Hepatobiliary – Pancreas (N = 4,832)	0.3 (15)	0.5 (10)
Musculoskeletal – Amputation (N = 4,800)	0.8 (37)	1.1 (29)
Esophagogastric – Gastric (N = 4,749)	0.3 (16)	0.7 (12)
Esophagogastric (N = 4,635)	0.0 (1)	0.1 (1)
Hysterectomy (N = 4,454)	0.1 (3)	0.2 (1)
Musculoskeletal – Arthroscopy (N = 4,255)	0.0 (0)	0.0 (0)
Musculoskeletal – Spine (N = 3,480)	0.1 (4)	0.3 (3)
Colorectal – Abdominoperineal resection (N = 3,169)	0.2 (7)	0.5 (5)
Musculoskeletal – Knee (N = 2,970)	0.1 (4)	0.2 (4)
Anorectal – Abscess (N = 2,508)	0.0 (0)	0.0 (0)
Simple skin and soft tissue (N = 2,383)	0.3 (6)	0.6 (4)
Colorectal – Low anastomosis (N = 2,293)	0.2 (4)	0.2 (2)
Hepatobiliary – Liver (N = 2,144)	0.3 (6)	0.8 (6)
Anorectal – Resection (N = 2,103)	0.0 (1)	0.0 (0)
Musculoskeletal – Fracture repair (N = 2,065)	0.1 (3)	0.3 (3)
Biopsy skin and soft tissue (N = 2,014)	0.1 (2)	0.2 (1)

Informed Consent

Perspectives on informed consent from patients in the U.K. suggest that major complications of surgery with an incidence of >1% should be discussed[15]; a majority of patients surveyed in a U.S. study suggests that rare but serious complications should also be discussed.[16] Mashour et al[1] showed that patients presenting for noncardiac, non-major-vascular surgery with any three or four of the risk factors listed in Table 2 have a 0.7% incidence of perioperative stroke and with five or more risk factors the incidence rises to 1.9%. It is therefore reasonable to discuss risk of perioperative stroke in patients with a history of stroke and other risk factors.

Recommendations

(1) Screen for risk factors of perioperative stroke, most notably remote or recent history of stroke, and communicate such risk to patients and providers (Category B, Level 2).

Timing of Surgery After Stroke

Patients with acute or recent stroke have impaired cerebrovascular autoregulation and chemoregulation for months,[17–20] rendering them dependent on systemic pressure and passive perfusion. This dependence creates particular risk for cerebral hypoperfusion, especially in the setting of general anesthesia and the physiologic perturbations of surgery (such as hemorrhage, anemia, hypotension). It has been suggested that elective surgery should be delayed from 1 to 3 months after a stroke in order to prevent a secondary cerebrovascular event.[21,22] To prevent perioperative stroke in patients with a history of recent cerebrovascular insult, it is likely beneficial to identify the cause of the initial stroke with investigations such as carotid imaging, magnetic resonance angiography, or echocardiogram. Known carotid disease should be treated based on current guidelines.[23,24]

Despite the intuition that delaying surgery after stroke is beneficial, a study of 173 surgical patients with a history of recent and remote stroke found no relationship between timing of stroke history and incidence of perioperative stroke.[21] One retrospective study of hip or knee replacement after stroke or acute coronary syndrome found that stroke within six months prior to surgery was not a predictor of postoperative mortality.[25] These findings were similar to a recent retrospective study of cardiac surgical patients; the time interval between stroke and coronary artery bypass graft surgery was not found to be a predictor of postoperative stroke or mortality.[26] Ultimately, the decision to proceed with surgery will always be a balance between the risks of perioperative stroke and the risks of delaying surgery further.

Recommendations

1. Discuss surgical timing with a neurologist and consider delaying elective surgical cases in patients with recent

TABLE 2. Independent Predictors of Perioperative Stroke Identified in Large Epidemiologic Studies

Predictors	Odds Ratio	Confidence Intervals
Independent Predictors found in Bateman et al, 2009. *Nationwide Inpatient Sample; hip, colon and lung surgery*		
Renal disease	2.98	2.52 to 3.54
Atrial fibrillation	1.95	1.69 to 2.26
History of stroke	1.64	1.25 to 2.14
Valvular disease	1.54	1.25 to 1.90
Congestive heart failure	1.44	1.21 to 1.70
Age (per 10 years)	1.43	1.35 to 1.51
Diabetes mellitus	1.18	1.01 to 1.39
Female (vs. Male)	1.21	1.07 to 1.36
Independent predictors found in Mashour et al, 2011. *American College of Surgeons- National Surgical Quality Improvement Program; broad population of noncardiac, nonvascular, nonneurologic surgery*		
Age $\geq$62 years	3.9	3.0 to 5.0
Myocardial infarction within 6 months	3.8	2.4 to 6.0
Acute renal failure	3.6	2.3 to 5.8
History of stroke	2.9	2.3 to 3.8
Pre-existing dialysis	2.3	1.6 to 3.4
Hypertension requiring medication	2.0	1.6 to 2.6
History of transient ischemic attack	1.9	1.3 to 2.6
Chronic obstructive pulmonary disease	1.8	1.4 to 2.4
Current smoker	1.5	1.1 to 1.9
Body mass index 35-40 kg/m^2 (protective)	0.6	0.4 to 0.9
Independent predictors found in Sharifpour, Moore et al, 2013. *American College of Surgeons- National Surgical Quality Improvement Program; noncarotid vascular surgery*		
Acute renal failure	2.03	1.39 to 2.97
History of stroke, transient ischemic attack, or hemiplegia	1.72	1.29 to 2.30
Female (vs. Male)	1.47	1.12 to 1.93
History of cardiac disease (myocardial infarction, congestive heart failure, angina, prior cardiac intervention)	1.42	1.07 to 1.87
Age (each additional year of life)	1.02	1.01 to 1.04

Note that certain variables relevant to stroke, such as atrial fibrillation and valvular disease, are not collected in the National Surgical Quality Improvement Program database.

TABLE 3. Possible Risk Classification for Bleeding According to Surgery/Procedure Type

Bleeding Risk Category	Type of Surgery/Procedure
High risk	Intracranial or spine surgery
	Major vascular surgery (aortic aneurysm repair, aortofemoral bypass)
	Major urologic surgery (prostatectomy, bladder tumor resection)
	Major orthopedic surgery (hip replacement)
	Lung resection
	Intestinal anastomosis surgery
	Permanent pacemaker or internal defibrillator placement
	Selected procedures: colonic polypectomy of large polyp, endoscopic retrograde cholangiopancreatography with sphincterotomy, kidney biopsy
Moderate risk	Other intra-abdominal surgery
	Other intrathoracic surgery
	Other orthopedic surgery
	Other vascular surgery
	Selected procedures: colonic polypectomy, prostate biopsy, cervical biopsy
Low risk	Laparoscopic cholecystectomy
	Laparoscopic inguinal hernia repair
	Noncataract ophthalmologic procedures
	Coronary angiography
	Gastroscopy or colonoscopy (with/without biopsy)
Very low risk (anticoagulation interruption not required)	Single tooth extraction
	Skin biopsy or selected skin cancer removal
	Cataract removal

Note that risk in certain cases may also relate to the consequences of bleeding (e.g., intracranial or spine procedure) rather than merely the volume of bleeding. Table reproduced with permission from Darvish-Kazem and Douketis, Perioperative management of patients having noncardiac surgery who are receiving anticoagulant or antiplatelet therapy: an evidence-based but practical approach. Semin Thromb Hemost 2012;38:652-660.

stroke until the etiology is investigated and the peak of autoregulatory disturbances has passed (likely at one month) (Opinion-based evidence, Category A). However, observational studies to date do not suggest a clear relationship between timing of past stroke history and incidence of postoperative stroke (Category B, Level 2).

Management of Anti-coagulants and Antiplatelet Drugs

There are two common clinical scenarios in which management of anticoagulants or antiplatelet drugs can be linked to risk of perioperative stroke. The first is management of anticoagulants for patients with atrial fibrillation, a major risk factor for perioperative stroke, and the second is the management of aspirin in patients with cardiovascular or cerebrovascular disease (e.g., for primary or secondary stroke prevention). The clinical dilemma relates to balancing the risks of excessive perioperative bleeding and the risk of rebound hypercoagulability in the setting of the prothrombotic state induced by surgery; a summary of surgical cases with various levels of expected blood loss can be found in Table 3.[27] There are few data to guide management of this situation that pertain specifically to perioperative stroke. The American College of Chest Physicians recommends that heparin therapy be considered for postoperative atrial fibrillation in patients with a history of stroke or transient ischemic attack[28]; an approach to the management of anticoagulation therapy can be found in Table 4.[27,29,30]

There are currently no studies of perioperative stroke and antiplatelet drug therapy for noncardiac, noncarotid surgery. Observational studies of cardiac surgery patients taking aspirin within 5 days prior to surgery (vs. not) revealed a protective effect of aspirin with respect to the outcome of perioperative stroke.[31,32] These studies are limited by an observational design but are consistent with a large randomized controlled trial finding a benefit to postoperative aspirin in preventing stroke after coronary artery bypass surgery.[33] It is as yet unclear how these data apply to noncardiac surgery, although recent studies in patients undergoing hip arthroplasty suggest

TABLE 4. Approach to Perioperative Bridging of Anticoagulation Management

Patient Group	Preoperative management	Postoperative management
Low-to-moderate bleeding risk	Stop therapeutic-dose LMWH bridging on morning (20-24 hours) before surgery Omit evening dose with twice-daily regimen Give 50% total dose with once-daily regimen	Resume therapeutic-dose LMWH bridging 24 hours after surgery and when there is adequate hemostasis
High bleeding risk	Stop therapeutic-dose LMWH bridging on morning (20-24 hours) before surgery Omit evening dose with twice-daily regimen Give 50% total dose with once-daily regimen	Resume therapeutic-dose LMWH bridging 48-72 hours after surgery and when there is adequate hemostasis Administer low-dose LMWH or avoid postoperative bridging

LMWH = low-molecular-weight heparin

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that aspirin reduces perioperative stroke.[34] In a nonoperative population, withdrawal of antiplatelet and antithrombotic medications was associated with a 5.2% incidence of stroke within 60 days of drug cessation.[35] Furthermore, nonoperative patients having strokes while off antiplatelet and antithrombotic drugs had greater morbidity and mortality compared to patients who continued taking them.[35] However, failing to stop these agents preoperatively may place patients at increased risk of intraoperative hemorrhage, which also increases the risk for perioperative stroke (see section on Intraoperative Recommendations).

Recently, the PeriOperative Ischemic Evaluation (POISE)-2 trial demonstrated that perioperative aspirin did not reduce the incidence of death or nonfatal myocardial infarction after noncardiac surgery, but did increase the risk of major bleeding.[36] Of note, patients who had aspirin therapy that was *initiated* in the course of the study had a reduced incidence of stroke compared to placebo; patients who were *continuing* aspirin therapy showed no reduction in stroke incidence. Stroke was not a primary outcome of the study and the authors acknowledged that the findings in the initiation group could be spurious. However, the results suggest that some patients at risk of stroke may benefit from preoperative initiation of aspirin therapy, but this must be balanced against the now well-documented risk of significant increases in bleeding and must be demonstrated in a larger trial.

Recommendations

(1) Medically manage atrial fibrillation and continue anticoagulation in patients with atrial fibrillation for minor surgeries or those in which high blood loss is unlikely. Discontinue anticoagulation in surgical patients at high risk of bleeding (with appropriate bridging strategies as indicated), but resume as soon as the risk of surgical bleeding is considered to be low (Opinion-based evidence, Category A).
(2) There is no evidence to suggest that continuation of aspirin in patients at risk for vascular complications reduces the risk of stroke after noncardiac surgery (Category A, Level 3).

Role of Preoperative Beta Blockers and Statins in Perioperative Stroke

The original POISE trial, which evaluated the cardioprotective effects of metoprolol in 8351 noncardiac surgery patients, demonstrated that patients receiving metoprolol had a significantly higher risk of stroke (hazard ratio 2.17, $p = 0.005$) and death (hazard ratio 1.33, $p = 0.032$).[37] Data from the POISE trial and other investigations contributed to a meta-analysis suggesting that beta blockers increase risk of nonfatal stroke after noncardiac surgery[38]; patients from the POISE trial constituted the majority in this study. A retrospective case-control study subsequent to the POISE trial suggested no increased risk of perioperative stroke with clinically routine doses of beta blockers[39]; a study of low-dose bisoprolol also concluded that there was no increased risk of perioperative stroke.[40] Based on these studies it is unclear if there is a drug-specific effect of metoprolol that increases risk of stroke or whether increased risk in the POISE trial was due to bradycardia and hypotension in the treatment group. Based on retrospective data from a single-center observational study, Mashour et al[8] demonstrated that routinely prescribed p.o. metoprolol conferred a higher risk of postoperative stroke compared to a matched cohort taking atenolol. These data are consistent with the results of a U.S.-wide Veterans Administration hospital study by London et al showing a higher risk of stroke after noncardiac surgery in patients taking metoprolol compared to atenolol.[41] In a single-center observational study, Ashes et al demonstrated that bisoprolol is associated with a lower stroke risk than either metoprolol or atenolol.[42] Current guidelines endorse the perioperative continuation of beta blockers in surgical patients who are already taking this class of drug. Large prospective studies are required to confirm that surgical patients who continue perioperative beta blockers are at increased risk of stroke if metoprolol is administered vs. another beta blocker. In surgical patients who are beta-blocker-naïve, high-dose beta blockers should not be administered without dose titration.[43]

As with beta blockers, discontinuation of statins in the perioperative period may have adverse consequences. With respect to nonoperative stroke, discontinuation of statins in individuals with acute ischemic stroke was associated with a high risk of early neurologic deterioration.[44] A recent, preliminary, retrospective study of asymptomatic surgical patients presenting for carotid endarterectomy suggested that statins could reduce neurologic injury, as defined by both stroke and cognitive dysfunction.[45] Statins have also been shown to reduce the incidence of atrial fibrillation and other adverse outcomes that may be associated

with postoperative stroke.[46,47] However, there are no data to suggest that starting statins in the preoperative period can prevent stroke around the time of noncardiac, nonneurologic and noncarotid surgery.

Recommendations

(1) Metoprolol or other beta blockers should only be started in the preoperative period with careful titration (Category A, Level 3).
(2) Continue beta blockers and statins throughout the perioperative period in patients already taking them (Opinion-based evidence, Category A with respect to stroke risk).

INTRAOPERATIVE RECOMMENDATIONS

Intraoperative events are frequently cited as a cause of postoperative stroke, despite controversial evidence. Stroke presenting in the postoperative period on the same day of surgery (which would suggest a clear intraoperative etiology) is relatively infrequent.[8] In this section we review the available data guiding intraoperative management of anesthetic technique, ventilation strategy, fluid and blood transfusion, glycemic control and blood pressure.

Anesthetic Technique

There has been extensive interest in the possible neuroprotective effects of anesthetic agents. Most studies have been in patients for whom cerebral ischemia is predictable; examples of predictable cerebral ischemia include carotid endarterectomy, cerebral aneurysm surgery and procedures requiring deep hypothermic circulatory arrest. For these procedures, anesthetic technique may be adjusted prospectively in an attempt to minimize neurologic injury. However, data supporting anesthetic neuroprotection even for these procedures is limited or absent. Given the low incidence of perioperative stroke in noncardiac and nonvascular surgical patients (making RCT difficult), investigators have studied neurologic outcome after cardiac surgery, in which the stroke risk is higher. Bilotta et al performed an extensive literature review of randomized trials but could not make definitive conclusions due to the small number and heterogeneity of studies.[48]

It is also important to consider anesthetics that might potentially increase stroke risk. For example, nitrous oxide is associated with an acute increase in plasma homocysteine concentrations, which could impair endothelial function and increase adverse cardiovascular events.[49] However, no association has been demonstrated in several large studies between intraoperative administration of nitrous oxide and postoperative stroke.[8,49–51]

A recent retrospective review of 57,000 patients[8] revealed no difference in stroke risk between regional and general anesthesia in noncardiac patients. However, a large database study (>200,000 patients) focusing on knee and hip arthroplasty found that neuraxial anesthesia was associated with a lower incidence of stroke (0.07%) compared to combined neuraxial/general anesthesia (0.12%) and general anesthesia (0.13%, $p = 0.006$).[52] Overall 30-day mortality was also reduced in the neuraxial (0.10; $n = 40{,}036$) and combined neuraxial-general (0.10; $n = 49{,}396$) groups compared to the general anesthesia group (0.18; $n = 292{,}804$; $p < 0.001$). Similarly, in a 2010 single center observational study of 18,745 consecutive joint replacements, general anesthesia was an independent predictor of postoperative stroke (OR 3.54, 95% CI 1.01-12.39).[53] In another patient population from the GALA trial, which studied carotid endarterectomy patients, there was no difference in stroke rates between general and regional anesthesia.[54] Thus, while there are no data in a broad, representative surgical population to support general vs. regional anesthetic technique, there are now at least two large outcomes studies suggesting that regional anesthesia for hip and knee arthroplasty may be associated with a lower risk of perioperative stroke.

Recommendations

1. Despite characteristics that would appear to predispose to perioperative stroke, nitrous oxide use has not been associated with an increased incidence of perioperative stroke (Category B, Level 1).
2. Recent retrospective data suggest that neuraxial techniques may be associated with a lower incidence of perioperative stroke for hip and knee arthroplasty (Category B, Level 1). No such data exist for other surgical populations.

Intraoperative Use of Beta Blockers

The most important intraoperative pharmacologic association with postoperative stroke is the administration of beta blockers. Mashour et al[8] retrospectively studied 57,218 noncardiac patients, of whom 55 had perioperative strokes. Intraoperative metoprolol administration was associated with a 3.3-fold increased risk of perioperative stroke ($p = 0.003$; 95% CI 1.4-7.8). No such association was found for intraoperative esmolol or labetolol. While the investigators also found intraoperative hypotension to be associated with perioperative stroke, no co-linearity existed between intraoperative metoprolol administration and hypotension.

The mechanism of metoprolol's apparent role in perioperative stroke is unclear. There is wide diversity in beta $_1$-adrenergic selectivity among beta antagonists, with bisoprolol having the greatest selectivity, atenolol intermediate and metoprolol the least selectivity among clinically used "cardioselective" beta blockers.[55] Recent animal data suggest that metoprolol, as a relatively nonselective $beta_1$ antagonist, may reduce brain tissue oxygenation by impairing $beta_2$ mediated cerebral vasodilation in mice.[56] Furthermore, in rats, metoprolol impairs the compensatory increase in cardiac output that occurs in response to anemia, reducing cerebral tissue oxygenation.[57]

Recommendations

1. Intraoperative metoprolol administration may place patients at increased risk for stroke (Category B, Level

1); these data have not been separately analyzed for patients taking metoprolol prior to surgery. Beta blockers with a short duration of action such as esmolol should be considered for intraoperative use (Opinion-based evidence, Category A).

Ventilation Strategies and Risk of Perioperative Stroke

There are limited data on the interaction of intraoperative $PaCO_2$ or $EtCO_2$ and stroke incidence. Any conclusion on ventilation strategies to reduce the risk of stroke is therefore speculative. There are, however, several considerations regarding ventilation and stroke: 1) Intraoperative hyperventilation has multiple systemic deleterious effects including: reduced lung compliance and potential for reduced oxygenation (from impaired V/Q mismatch and increased shunt); increased myocardial oxygen demand and reduced supply (coronary vasoconstriction); potential dysrhythmias; and reduced cerebral blood flow; 2) Although in theory hyperventilation has the potential to favorably redistribute flow from normal brain to relatively ischemic regions ("inverse steal phenomenon"), in animals subjected to middle cerebral artery occlusion this does not happen and in fact there may be an increase in the size of the region at risk for stroke.[58–61] 3) Nonoperative stroke patients who were hypocapnic had worse outcomes compared to normocapnic patients.[62] Until more intraoperative data are available, we are unable to make any conclusions regarding whether hypocapnia places patients at risk for perioperative stroke or whether there are ventilation strategies to reduce the risk of stroke. It seems likely that hypocapnia, because of the reduction in cerebral blood flow, is undesirable in patients with risk factors for perioperative stroke and may be one of several factors that negatively affect outcome in those patients who suffer stroke perioperatively.

Recommendations

1. There are no data on which to base recommendations regarding intraoperative ventilation strategies and the incidence of perioperative stroke (Category D, Silent).

Intraoperative Hemorrhage and Blood Transfusion Therapy

There is evidence linking intraoperative hemorrhage and anemia to postoperative stroke, particularly in cardiac patients. It is difficult to determine whether absolute hematocrit (and thus oxygen carrying capacity), the volume of hemorrhage itself (suggestive of surgical complexity), hypotension and/or physiologic stress accounts for this association with perioperative stroke. In control-matched cardiac surgery patients suffering postoperative stroke, Bahrainwala et al.[63] demonstrated that both post-cardiopulmonary bypass hemoglobin level and the volume of blood transfused intraoperatively were independent predictors of stroke. The number of units transfused was directly associated with the risk of stroke and, although transfusion attenuated the relationship between post-bypass hemoglobin and stroke, it did not eliminate risk.

In a retrospective study of 651,775 patients undergoing noncardiac, noncarotid and nonneurologic surgery, patients receiving more than four units of packed red blood cells (as a surrogate for major hemorrhage) had an approximately 2.5 fold increased risk for stroke or Q-wave myocardial infarction.[64] The POISE trial also found "significant bleeding" to be an independent predictor of postoperative stroke (adjusted OR 2.18, 95% CI 1.45-8.52) in the population of high risk patients undergoing noncardiac surgery.[37] The authors commented on a dose-response relationship between the volume transfused and stroke but did not examine the interaction between hypotension and anemia. There is evidence in nonoperative stroke that anemia is associated with stroke in the absence of hypotension,[65,66] and thus it seems reasonable to assume that this association exists in surgical patients as well.[67,68] Furthermore, recent observational data suggest that intraoperative anemia (hemoglobin < 9 gm/dl) in the setting of beta blockade increases risk of stroke, but the study was not able to distinguish acute from chronic use.[42]

Recommendations

1. For noncardiac, nonneurologic surgical patients already taking a beta blocker, a hemoglobin < 9.0 gm/dl should be avoided in order to minimize risk of stroke (Category B, Level 1).

Glucose Management

Hyperglycemia has long been recognized to have a negative influence on neurologic outcomes following cerebral ischemia.[69] This is true for both global and focal ischemia as well as specific surgical populations for which focal ischemia is predictable, including cardiothoracic surgery,[70] carotid endarterectomy[71] and open cerebral aneurysm ligation.[72] It should also be noted that in a relatively small randomized trial of cardiac patients, intensive intraoperative insulin therapy (glucose goal of 80-100 mg/dL) was associated with an increased risk of stroke and death, despite no increases in hypoglycemic events.[73] A recent meta-analysis concluded that intravenous insulin for tight glycemic control within 24 hours after nonoperative stroke may not be beneficial and may increase the risk of hypoglycemia.[74] This is clearly a complex issue and most authors recommend an intervention with serum glucose exceeding 150 mg/dL, with an absolute upper limit of 180 mg/dL, in hyperglycemic patients undergoing major surgery.[75,76]

Recommendations

1. In patients at high risk for perioperative stroke undergoing surgery, glucose monitoring is recommended, with a target range of 60-180 mg/dL (Opinion-based evidence, Category A).

Blood Pressure Management

Intraoperative hypotension is frequently cited as a cause of postoperative stroke. Until recently, however, there

was very little evidence to support this belief and even the neurology literature concluded that, "Ischemic strokes after general surgery commonly occur after an asymptomatic interval…Hypotension rarely accounts for postoperative strokes."[11] Studying the effect of blood pressure has proven difficult given the diversity of definitions and the very low incidence of perioperative stroke, despite the very high incidence of intraoperative hypotension.[77] The 2008 POISE study raised concern regarding the potential role of metoprolol in perioperative stroke but also re-focused attention on the possible role of intra- and postoperative hypotension in its pathogenesis.[37] Patients receiving metoprolol had an increased perioperative risk of clinically significant hypotension (HR 1.55, 95% CI 1.38-1.74), which in turn was associated with death and stroke (OR 4.97 95% CI 3.62-6.81). More recently, Bijker et al[78] conducted a retrospective case-control study reviewing 48,241 consecutive noncardiac and nonneurosurgical patients and found an overall stroke incidence of 0.09% within 10 days of surgery. They determined that a decrease in intraoperative mean arterial pressure of more than 30% below baseline was associated with postoperative stroke. However, the odds ratio of 1.013 reveals an effect size of unclear clinical significance. The authors posit that unrecognized or poorly quantified hypotension in the postoperative period may be more important to stroke genesis than hypotension during the highly monitored and regulated intraoperative period and suggest that intraoperative hypotension may be predictive of postoperative hemodynamic instability rather than an independent cause of postoperative stroke. They furthermore conclude that "hypotension is best defined as a decrease in mean blood pressure relative to a preoperative baseline, rather than an absolute low blood pressure value." Mashour et al[8] also found that, for median values in a 10-minute epoch, a $<20\%$, $<30\%$ or $<40\%$ decrease in mean arterial or systolic blood pressure is associated with stroke. Causal relationships were not investigated in this study.

In addition to the timing of perioperative stroke, the low incidence of watershed infarctions in noncardiac surgical patients[79] also argues against intraoperative hypotension as a defining risk for postoperative stroke. This is further complicated by the controversy of whether watershed infarcts are indeed produced by systemic hemodynamic factors or local microemboli. Cortical watershed infarcts are probably embolic in nature, especially if the large cerebral conducting vessels are atheromatous, whereas deep white matter infarcts are probably of hemodynamic origin.[80,81]

Finally, postural hypotension may also play a role in stroke after noncardiac surgery. In 2005, Pohl and Cullen reported four cases of ischemic brain and spinal cord injury after shoulder surgery in the beach chair position.[82] Since that time, significant attention has been given to the risk of unrecognized reduced cerebral perfusion pressures and resultant neurologic injury in the beach chair position. Neurologic injuries have occurred in the beach chair position at recorded blood pressures that most anesthesiologists would consider acceptable.[83] Assuming a 0.8 mm Hg decrease in MAP for every 1 cm gradient, and a 15-30 cm gradient between the brachial artery (cuff measurement) and the brainstem, the MAP at the level of the brainstem could be 12-24 mm Hg lower than the pressure measured by cuff on the nonoperative arm. Clearly this relationship is greatly worsened if the cuff is placed on the lower extremity[84]; this differential should also be kept in mind when leveling a transducer if invasive blood pressure monitoring is used. There are numerous studies using near infrared spectroscopy that document a high incidence of desaturations measured in the beach chair position.[85–87] The devices are intended to measure saturation in a volume of tissue comprising neural cells, arterioles and veins. A reduction in saturation may represent cellular oxygen desaturation but may also represent a change in the ratio of arterial to venous blood. The latter may occur with vasopressors or head positioning that limits cerebral venous drainage. However, it is important to note that the degree and duration of cerebral desaturation required to produce neurologic injury is also not yet known but a reduction of $>20\%$ is thought to be cause for concern.[88,89] Some orthopedic surgeons still support the safety of induced hypotension in the beach chair position.[90] However, given the frequency of severe hypotension and electroencephalographic changes consistent with cerebral ischemia (6%), this technique cannot be endorsed.

In sum, it seems that there is an association between intraoperative hypotension and perioperative stroke, but the clinical significance of this association is poorly defined. Further prospective research is clearly necessary to make strong evidence-based recommendations regarding intraoperative blood pressure management. It is probable that there are subsets of patients at increased risk for stroke who clearly are harmed by hypotension or low flow states. Some of these populations may include patients with unrecognized critical stenosis of carotid or intracranial arteries or congenital anomalies of the cerebral circulation.[91]

Recommendations

1. There are data to support an association between intraoperative hypotension and perioperative stroke (Category B, Level 1). As such, intraoperative hypotension should be avoided in patients at high risk of perioperative stroke (Opinion-based evidence, Category A).
2. Intraoperative hypotension should be defined as a percent reduction from baseline blood pressure rather than an absolute value (Category B, Level 1).
3. For surgery in the beach chair position, noninvasive blood pressure measurement by cuff should always be performed on the nonoperative upper arm (as opposed to lower extremity) and consideration should be given to the blood pressure gradient between the brachial artery and brain (Opinion-based evidence, Category A). Induced hypotension for shoulder surgery in the beach chair position should always be approached with caution, especially in patients at risk for stroke (Opinion-based evidence, Category A).

POSTOPERATIVE RECOMMENDATIONS

Stroke Team, Networks, and Communication

In the event of an acute stroke, preventing secondary injury and achieving optimal outcome are based on rapid recognition, communication and management. Evaluation and treatment of acute perioperative stroke, which is typically ischemic, should be consistent with the most recent American Heart Association Guidelines for the Management of Patients with Acute Ischemic Stroke.[92] The advantage of an acute stroke response algorithm is self-evident, since the activities of multiple personnel must be coordinated efficiently among practitioners in fields such as anesthesiology, neurology, radiology, and interventional neuroradiology. An institutional pathway promotes higher efficiency, more rapid therapeutic intervention, and a more reliable transfer of information pertinent to the patient's optimal care[93]

Recommendations

1. An organized protocol for emergency evaluation of surgical patients with suspected perioperative stroke is recommended (Category A, Level 3).

Assessing Stroke

There is a need to develop and evaluate simple, quick screening tools for stroke that can be used in the perioperative period. Such tools need to be suitable for preoperative screening as well as routine use by nursing staff on surgical wards or intensive care units. Examples of such tests that may be suitable for postoperative use include the Face Arm Speech Time (FAST),[94] Los Angeles Prehospital Stroke Screen (LAPSS),[95] Melbourne Ambulance Stroke Screen (MASS),[96] and Recognition of Stroke in the Emergency Room (ROSIER).[97]

Various physiologic, pharmacologic and pathologic factors in the postanesthetic period can mask symptoms of intraoperative or postoperative stroke. Careful evaluation and resolution of pharmacologic contributions to a neurologic deficit is paramount to reduce delay in stroke diagnosis.[98] Certain drugs that are commonly used during the intraoperative period can be reversed, including narcotics (with naloxone), benzodiazepines (with flumazenil), and neuromuscular blockers (with a cholinesterase inhibitor or sugammadex, where available). Although the presence of residual inhaled anesthetics can be quantified routinely in the operating room at the end of a surgical case, the effect of drugs such as propofol and dexmedetomidine cannot be conveniently measured in real time.

Delayed emergence, altered mental status and/or the presence of new focal neurologic deficit in the absence of convincing confounders should raise suspicion for stroke.[99] A detailed neurologic examination should include the use of the National Institutes of Health Stroke Scale (NIHSS) (Table 5; see also: http://www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf or http://www.ninds.nih.gov/doctors/NIH_Stroke_Scale_Booklet.pdf). Clinical evaluation also includes measurement of blood pressure, oxygen saturation, temperature, blood glucose, serum electrolytes, complete blood count and coagulation status. Immediate diagnostic studies of all patients with suspected stroke should include noncontrast computed tomography (CT) or magnetic resonance imaging (MRI) of the brain to determine whether the stroke is ischemic or hemorrhagic in origin and to correlate neurologic deficit with radiologic findings.[100] The role of CT angiography and CT perfusion imaging is to provide supplementary information, especially in determining the need for urgent endovascular intervention. The presence of a large ischemic penumbra or thrombus in a major vessel (e.g., middle cerebral artery) would prompt consideration of endovascular therapy. One

TABLE 5. National Institutes of Health Stroke Scale

Tested Item	Title	Response and Scores
1A	Level of consciousness	0—Alert 1—Drowsy 2—Obtunded 3—Coma/unresponsive
1B	Orientation questions (2)	0—Answers both questions correctly 1—Answers 1 question correctly 2—Answers neither question correctly
1C	Response to commands (2)	0—Performs both tasks correctly 1—Performs 1 task correctly 2—Performs neither task correctly
2	Gaze	0—Normal horizontal movements 1—Partial gaze palsy 2—Complete gaze palsy
3	Visual fields	0—No visual field defect 1—Partial hemianopia 2—Complete hemianopia 3—Bilateral hemianopia
4	Facial movement	0—Normal 1—Minor facial weakness 2—Partial facial weakness 3—Complete unilateral palsy
5	Motor function (arm) a. Left b. Right	0—No drift 1—Drift before 5 seconds 2—Falls before 5 seconds 3—No effort against gravity 4—No movement
6	Motor function (leg) a. Left b. Right	0—No drift 1—Drift before 5 seconds 2—Falls before 5 seconds 3—No effort against gravity 4—No movement
7	Limb ataxia	0—No ataxia 1—Ataxia in 1 limb 2—Ataxia in 2 limbs
8	Sensory	0—No sensory loss 1—Mild sensory loss 2—Severe sensory loss
9	Language	0—Normal 1—Mild aphasia 2—Severe Aphasia 3—Mute or global aphasia
10	Articulation	0—Normal 1—Mild dysarthria 2—Severe dysarthria
11	Extinction or inattention	0—Absent 1—Mild (loss, 1 sensory modality lost) 2—Severe (loss, 2 modalities lost)

of the limitations of conventional MRI is the duration of the scan; however, diffusion-perfusion MRI enables the identification of an ischemic penumbra and identification of patients who may benefit from late thrombolysis.[101]

Recommendations

1. Use of a stroke rating scale, preferably the NIHSS, is recommended (Category A, Level 3).
2. Emergency imaging of the brain is recommended before initiating any specific therapy to treat acute postoperative stroke (Category A, Level 1).

Acute management of ischemic stroke

In considering the management of acute perioperative stroke, it will be helpful to apply, where appropriate, therapeutic strategies for ischemic stroke treatment in nonsurgical settings. Ischemic stroke is the most common form of perioperative stroke and thus the present recommendations are directed at its treatment. Identification of a hemorrhagic stroke with head CT in the surgical patient will prompt a distinct pathway of investigation and management, with distinct recommendations for blood pressure control.[102,103]

Interventions such as recombinant tissue plasminogen activator (rtPA) should be considered with a multidisciplinary team that includes stroke neurology, interventional neuroradiology and the primary surgical service. The risk/benefit balance will vary depending on the patient, severity and location of the stroke, and type of surgical intervention.

Intravenous rtPA is the drug of choice for thrombotic strokes, but it is contraindicated in a number of situations (Table 6). Importantly, major surgery—with the exception of intracranial or spinal surgery—is a relative rather than absolute contraindication, but there is a paucity of literature regarding the management of stroke after a noncardiac, nonvascular or nonneurologic procedure. Oral administration of aspirin (initial dose is 325 mg) within 24 to 48 hours after stroke onset is recommended but is not a substitute for other acute interventions such as rtPA.[92] Furthermore, the administration of aspirin (or other antiplatelet agents) as an adjunctive therapy within 24 hours of intravenous fibrinolysis is not recommended.[92] Urgent anticoagulation with the goal of preventing early recurrent stroke, halting neurological deterioration or improving outcomes after ischemic stroke is not recommended,[92] nor is the initiation of anticoagulation therapy within 24 hours of treatment with intravenous rtPA. In addition to intravenous rtPA, intraarterial thrombolysis has American Heart Association Class IIa, Level C evidence of safety and efficacy.[100,104] Although endovascular mechanical thrombolysis has recently been shown not to be superior to intravenous rtPA,[105] it may be an attractive alternative in the postoperative patient. Again, all interventions should be discussed with a multidisciplinary team including neurologists, neuroradiologists, and the primary surgical service, while keeping in mind that "time is brain."

The initial treatment of stroke is usually best achieved in a subspecialty acute care setting such as a neurocritical care or stroke unit. Maintaining appropriate physiologic stability is critical during acute stroke care. Hypoxemia is associated with poor neurologic/ outcomes and should therefore be monitored with pulse oximetry; supplemental oxygen should be used to maintain SpO_2 saturation greater than 94%.[92] The airway should be secured in patients with depressed levels of consciousness (Glasgow Coma Scale <8), signs of brainstem dysfunction, or inability to protect the airway. Tracheal intubation and mechanical ventilation may also be helpful in the management of increased intracranial pressure or for those who have suspected malignant brain edema.

Patients with acute cerebral ischemia should have cardiac monitoring for at least the first 24 hours, and any serious arrhythmia should be treated.[92] Myocardial ischemia and cardiac arrhythmias are potential sequelae of acute cerebrovascular disease and systemic hypertension is common after stroke. Common postoperative conditions that may contribute to hypertension include stress response to surgery, pain, nausea, hypervolemia, full bladder, or physiological response to hypoxia. Every effort should be made to preserve cerebral perfusion pressure during the first 24

TABLE 6. Inclusion and Exclusion Criteria for Patients with Acute Ischemic Stroke who could be Treated with Intravenous rtPA within Three Hours from Symptom Onset

Inclusion criteria
Diagnosis of ischemic stroke causing measurable neurologic deficit
Onset of symptoms <3 hours before beginning treatment
Aged ≥ 18 years
Exclusion criteria
Significant head trauma or prior stroke in previous 3 months
Symptoms suggest subarachnoid hemorrhage
Arterial puncture at noncompressible site in previous 7 days
History of previous intracranial hemorrhage
Intracranial neoplasm, arteriovenous malformation, or aneurysm
Recent intracranial or intraspinal surgery
Elevated blood pressure (systolic >185 mm Hg or diastolic >110 mm Hg)
Active internal bleeding
Acute bleeding diathesis
Platelet count $<100\,000/mm^3$
Heparin received within 48 hours, resulting in abnormally elevated aPTT greater than the upper limit of normal
Current use of anticoagulant with INR >1.7 or PT >15 seconds
Current use of direct thrombin inhibitors or direct factor Xa inhibitors with elevated sensitive laboratory tests
Blood glucose concentration <50 mg/dL (2.7 mmol/L)
Computed tomography demonstrates multilobar infarction (hypodensity $>1/3$ cerebral hemisphere)
Relative exclusion criteria
Recent experience suggests that under some circumstances—with careful consideration and consideration of risk/benefit ratio—patients may receive fibrinolytic therapy despite 1 or more relative contraindications. Consider risk to benefit of intravenous rtPA administration carefully if any of these relative contraindications are present:
Only minor or rapidly improving stroke symptoms (clearing spontaneously)
Pregnancy
Seizure at onset with postictal residual neurological impairments
Major surgery or serious trauma within previous 14 days
Recent gastrointestinal or urinary tract hemorrhage (within previous 21 days)
Recent acute myocardial infarction (within previous 3 months)

hours. Unless the patient is eligible for acute reperfusion intervention, systolic blood pressure is usually treated only if it is greater than 220 mm Hg, and diastolic pressure is treated only if it is greater than 120 mm Hg.[92] In patients who receive rtPA (intravenous or intra-arterial) or undergo mechanical clot retrieval, systolic blood pressure above 180 mm Hg and diastolic pressure above 105 mm Hg should be treated with anti-hypertensive drugs such as labetolol or nicardipine.[92] However, no data are available to guide selection of medications for the lowering (or raising) of blood pressure in the setting of acute ischemic stroke. Both hypovolemia and unstable cardiac arrhythmias may result in arterial hypotension, which is detrimental in the setting of stroke. As such, correction of hypovolemia with normal saline and restoration of normal sinus rhythm is beneficial.[92]

Recommendations

1. If not contraindicated based on multidisciplinary discussion regarding risks of hemorrhage, intravenous/intra-arterial rtPa or mechanical thrombolysis should be considered as soon as possible for the surgical patient with an acute ischemic stroke (Opinion-based evidence, Category A).
2. In patients who receive rtPA (intravenous or intra-arterial) or undergo mechanical clot retrieval, systolic blood pressure above 180 mm Hg and diastolic pressure above 105 mm Hg should be treated with anti-hypertensive drugs such as labetolol or nicardipine (Category A, Level 1).
3. The administration of aspirin (or other antiplatelet agents) as an adjunctive therapy within 24 hours of intravenous fibrinolysis is not recommended (Opinion-based evidence, Category A).
4. Supplemental oxygen should be provided to maintain oxygen saturation >94% (Opinion-based evidence, Category A).
5. Airway support and ventilator assistance are recommended for treatment of patients with decreased consciousness or bulbar dysfunction that causes compromise of respiration (Opinion-based evidence, Category A).
6. Baseline electrocardiogram and troponin assessment is recommended (Category A, Level 3).

CONCLUSIONS

Perioperative stroke can be a devastating adverse outcome for surgical patients and their families and further research is clearly required. Recognition of patients at high risk for stroke during or after noncardiac, nonneurologic surgery is critical. Consistent risk factors include advanced age, renal disease and history of stroke or transient ischemic attack. Continuation of beta blockers and statins is important for prevention; if indicated, beta blockers should only be started in the perioperative period with careful titration. Recent evidence suggests that continuation of aspirin in patients at risk of stroke after noncardiac surgery is not indicated and may increase bleeding risk. Intraoperative hypotension should be avoided in surgical patients at high risk of perioperative stroke and for those in the beach chair position. Postoperative vigilance and appropriate stroke screening for high risk surgical patients is prudent. For surgical patients manifesting symptoms or signs of stroke, timely neurology/stroke consultation and emergent neuroimaging are essential. Major noncardiac, nonneurologic surgery is not an absolute contraindication to intravascular administration of rtPa; mechanical thrombolysis is also an option for those at high risk of surgery-related hemorrhage.

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