

Risky Cerebrovascular Anatomic Orientation: Implications for Brain Revascularization

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Key words

- Anterior cerebral artery
- Cerebral ischemia
- Cerebral watershed infarction
- Extracranial (EC)-Intracranial (IC) bypass
- Revascularization
- STA-ACA bypass graft

Abbreviations and Acronyms

- ACA:** Anterior cerebral artery
CM: Callosomarginal artery
CT: Computed tomography
DSA: Digital subtraction angiography
MCA: Middle cerebral artery
OA: Occipital artery
PC: Paracentral artery
PIFA: Posterior internal frontal artery
POD: Postoperative day
Ra: Recipient artery
SPECT: Single-photon emission computed tomography
STA: Superficial temporal artery
TIA: Transient ischemic attack

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INTRODUCTION

Besides the rarity of isolated anterior cerebral artery (ACA) occlusion, its revascularization remains a surgical challenge.^{1,2} Surgical options, including “intracranial (IC)-IC”^{1,3} or “extracranial (EC)-IC with or without graft”^{2,4-8} bypass, need to be individualized for each patient.

This study documents a risky vascular anatomic orientation that might play an important role in the postoperative hemodynamics following anterior cerebral artery (ACA) revascularization. A 71-year-old woman presented with uncontrollable frequent right lower limb transient ischemic attacks (TIAs) attributed to a left cerebral ischemic lesion due to severe left ACA stenosis. She underwent successful left-sided superficial temporal artery–ACA bypass using interposed vascular graft. The patient awoke satisfactory from anesthesia; however, on postoperative day 1, she developed right-sided hemiparesis. Extensive postoperative investigations disclosed that watershed shift infarction was considered the etiology for this neurologic deterioration.

Controversy remains concerning the effectiveness of ACA revascularization procedures regarding prevention of strokes² despite successful results.^{1-4,6-8} To the best of our knowledge, this report represents the first study that highlighted watershed infarction following ACA revascularization, proposing risky vascular anatomic etiology and discussing better options.

CASE REPORT

History and Examination

In 2010, a 71-year-old woman was referred to our hospital because of frequent transient ischemic attacks (TIAs), in the form of right-sided predominant lower limb motor weakness, despite medical treatment (clopidogrel for 1-month duration). She was examined with magnetic resonance imaging, computed tomography (CT) angiography, digital subtraction angiography (DSA), and single-photon emission CT (SPECT). Those studies revealed a small, left, high-parietal ischemic lesion, severe stenosis of the left ACA, mild stenosis of the middle cerebral artery (MCA), insufficient collateral flow (Figure 1A–D), and left-sided areas of cerebral hypoperfusion (Figure 2).

Preoperative Planning

Following 1 week of intravenous heparinization, EC-IC bypass with occipital artery (OA) vascular graft was tailored for this patient. Other procedures, including

endovascular management or indirect bypass coupled with medication, were considered risky and potentially ineffective in our atherosclerotic patient with severe A2 stenosis.

Operation

She was positioned prone-lateral, and her head was fixed with a 4-pin Sugita head holder. Doppler flowmeter was used to map the parietal branch of the superficial temporal artery (STA) and OA. The skin incision was made along the course of the parietal branch and extended upward to the midline and then along the midline downward to the bregma (Figure 3A and B). The medial frontal craniotomy was performed. The dura was opened, and the posterior internal frontal artery (PIFA) (0.9 mm in size) was selected as the recipient (Figure 3C). This arterial flow was weak. Indocyanine green videoangiography showed slow antegrade flow of ACA. A free OA graft (5 cm in length, 1.0 mm in diameter) was harvested after placing a linear skin incision. The parietal branch of STA (1.5 mm) was prepared for anastomosis. End-to-end STA-OA anastomosis was done with a total of 10 stitches (Figure 3D). End-to-side OA-PIFA anastomosis was achieved with 9 stitches of 10-0 nylon, including 2 stay sutures (Figure 3E). The occlusion time was 39 minutes. Patency of the anastomosis was confirmed with Doppler flowmeter and indocyanine green videoangiography.

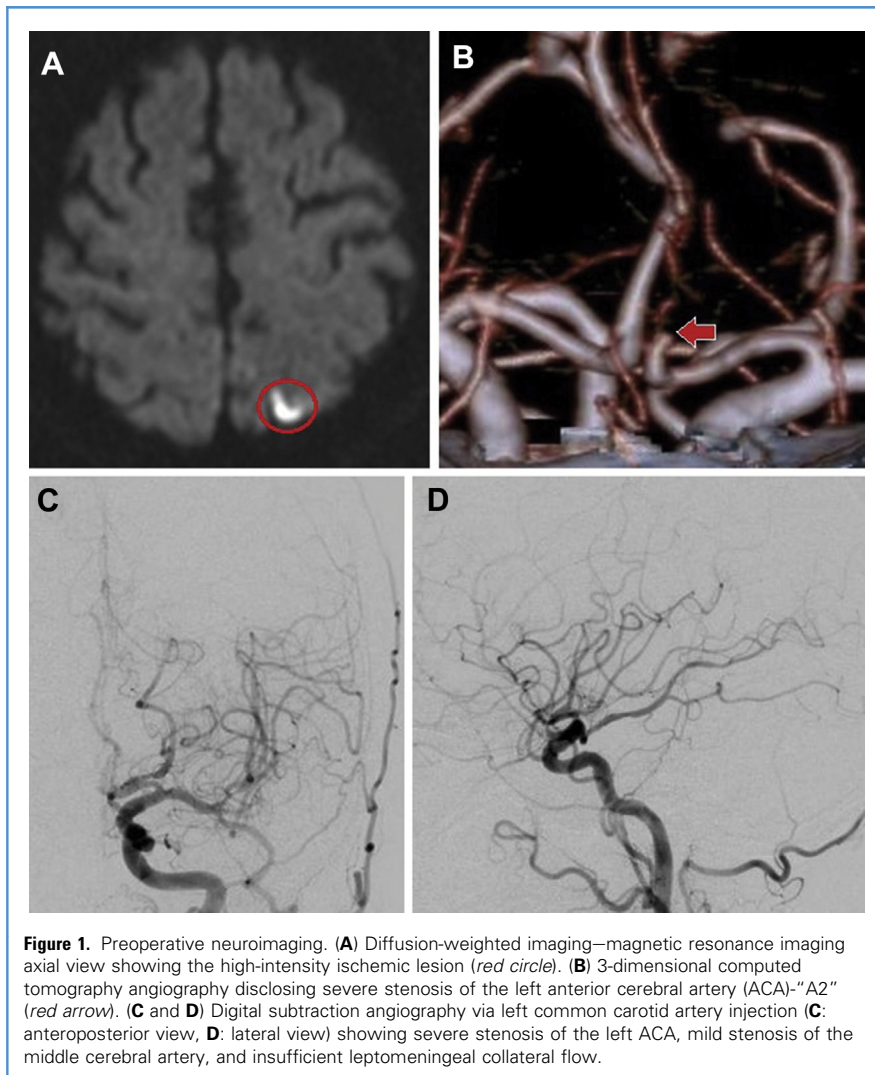


Figure 1. Preoperative neuroimaging. (A) Diffusion-weighted imaging—magnetic resonance imaging axial view showing the high-intensity ischemic lesion (red circle). (B) 3-dimensional computed tomography angiography disclosing severe stenosis of the left anterior cerebral artery (ACA)—“A2” (red arrow). (C and D) Digital subtraction angiography via left common carotid artery injection (C: anteroposterior view, D: lateral view) showing severe stenosis of the left ACA, mild stenosis of the middle cerebral artery, and insufficient leptomeningeal collateral flow.

Postoperative Course and Follow-up

She awoke from anesthesia without any neurologic deficits. Immediate postoperative CT excluded iatrogenic complications. No further TIAs developed after surgery. However, on postoperative day (POD) 1, she developed sudden right-sided hemiparesis. Urgent CT and magnetic resonance imaging (Figure 4A) disclosed worsening of the baseline ischemic lesion. Aspirin and cilostazol were administered immediately. Postoperative MR angiography on POD1 exhibited patency of the anastomotic site and its territories (Figure 4B). Persistent patency of the bypass was confirmed with DSA (Figure 4C and D) on POD3. Postoperative SPECT disclosed increased cortical uptake and confirmed watershed

shift (Figure 5). The patient was referred for rehabilitation, and her symptoms did not resolve at the time of discharge.

DISCUSSION

Despite marked advances in recent revascularization techniques and perioperative treatment, some patients suffer cerebral infarction in the early postoperative period.^{9,10}

ACA Revascularization Procedure

In order to perform successful surgery in our high-risk patient and to overcome additional risk factors for postoperative neurologic worsening,² we studied our options for ACA bypass¹⁻⁸ and their limitations and then took great

consideration during graft selection.^{7,10-12}

Although the carotid occlusion surgery study¹³ reported a 15% risk of perioperative strokes in patients undergoing STA-MCA bypass for chronic ischemia, the subemergent ischemia and the necessity for ACA revascularization in our patient seemed irrelevant to the Carotid Occlusion Surgery Study inclusion criteria. Accordingly, the STA-OA-PIFA bypass (Figure 6A and B) was considered the best option.

Postoperative Watershed Infarctions

Watershed shift-induced cerebral ischemia can lead to transient or permanent postoperative neurologic deteriorations.^{6,9,14-21} Interestingly, postoperative infarction in patients from Asian countries (as our patient) showed a more aggressive clinical course.⁹

The neurologic deterioration in the early postoperative period is referred to a focal decrease in cerebral perfusion⁹ remote from the anastomotic site (Figure 6C). Perioperative SPECT studies detected these focal changes (see Figures 2 and 5). Herein, extensive postoperative investigations ruled out all intraoperative events, including hypotension,¹⁸ as the patient awoke satisfactory and deterioration occurred on the next day. Also, clear-cut causes (as intracranial hematomas) were also excluded by the early postoperative CT. Additionally, on the basis of postoperative neuroimaging, obvious causes (occlusion or vascular spasm) of neurologic deterioration were ruled out. The region adjacent to the revascularization site showed an increased uptake on SPECT (see Figure 5), MR angiography exhibited a marked increase in signal intensity of the anastomosed vessel (see Figure 4B), and patent STA-OA-PIFA bypass was confirmed (see Figure 4C and D). Embolic source arising from the anastomotic site was also excluded on the basis of the infarction pattern. Accordingly, a new reversing flow pattern (see Figure 6C), after bypass grafting,¹⁵ induced significant hypoperfusion in the remote ACA territory and was considered the key for the postoperative neurologic deterioration in our patient. This was supported by the following: 1) Our patient was at a very high risk for decreased focal perfusion⁹ due to unstable hemodynamic^{7,21} as she presented with

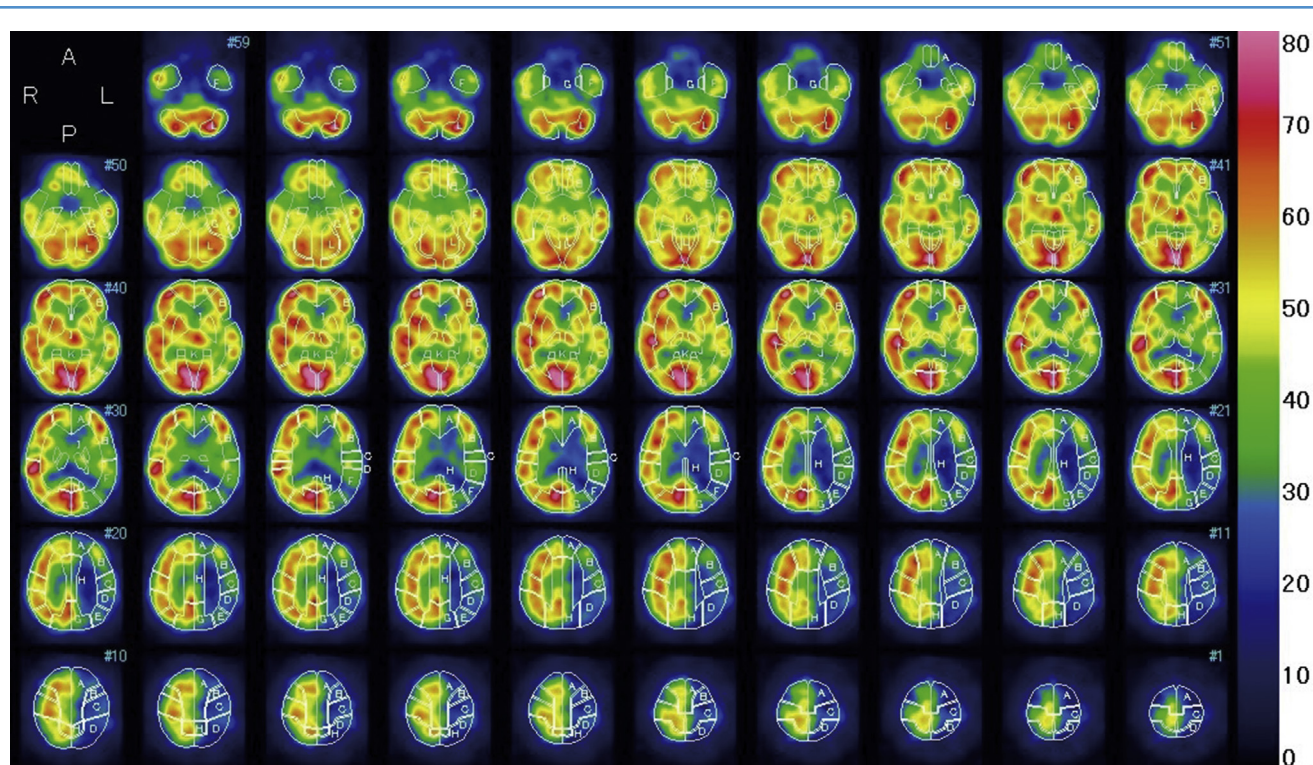


Figure 2. Preoperative resting ^{123}I -iodoamphetamine single-photon emission computed tomography study, revealing left-sided regions of

hypoperfusion. Colors represent perfusion levels according to the color scale on the right.

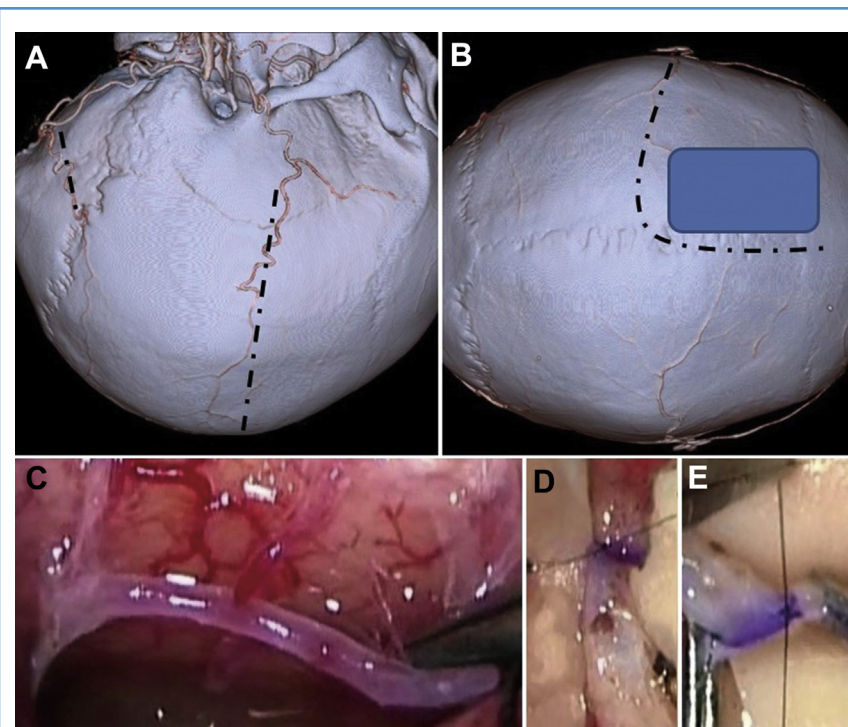


Figure 3. Operative procedure. (A) and (B), 3-dimensional computed tomography angiography showing the correlation between intraoperative head orientation, targeted vessels, locations of the skin incisions (dashed lines), and the craniotomy (blue box). (C, D, and E), Intraoperative microscopic video-captured images. (C) The posterior internal frontal artery (PIFA) was selected as a recipient. (D) Starting the end-to-end superficial temporal artery-occipital artery (OA) graft anastomosis. (E) End-to-side OA graft-PIFA anastomosis (toe suture).

frequent TIAs (3–4 attacks/day) and preoperative cerebral infarction (see [Figure 1A](#)) during the month preceding surgery; 2) this watershed shift contributed to neurologic deterioration in the early postoperative period; and 3) the perioperative SPECT studies detected a distant focal decrease in cerebral perfusion^{9,16} (see [Figure 5](#)).

Vascular Anatomic Orientation. Reviewing the microsurgical anatomy of the distal ACA²² revealed that the relationship of its terminal branches and their origins is greatly variable. This 3D vascular anatomic orientation might have a great role in determining the postoperative hemodynamics ([Figure 7A](#) and [B](#)). Herein, the recipient artery “PIFA” was almost in a straight angle with the parent artery “callosomarginal artery (CM).” This made the new retrograde blood flow “after bypass” and the normal antegrade blood flow facing each other directly. Subsequently, watershed shift occurred due to hemodynamic changes and reversal of flow at a specific point “branch point of paracentral artery.” This

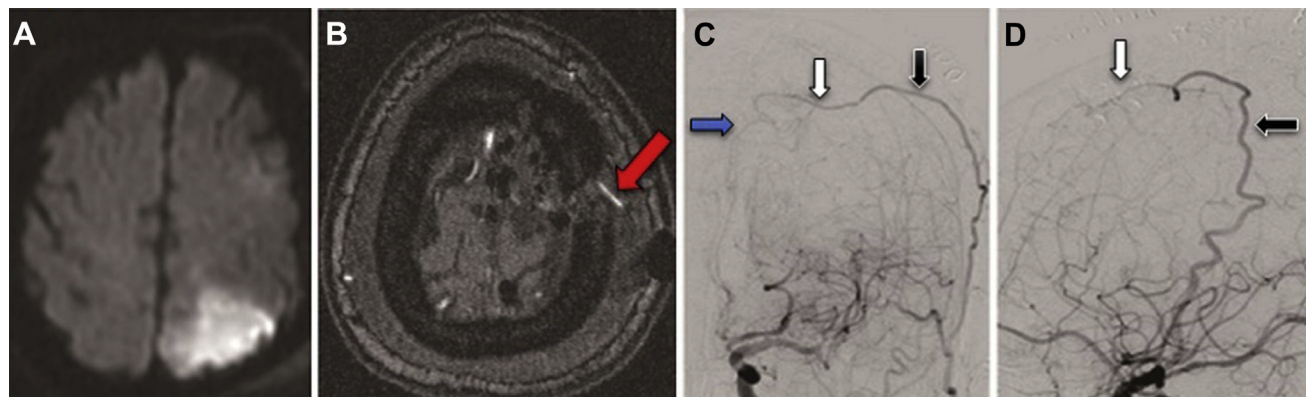


Figure 4. Postoperative neuroimaging. **(A)** Diffusion-weighted imaging—magnetic resonance (MR) imaging axial view “POD 1” showing left high parietal watershed ischemic stroke. **(B)** MR angiography on POD 1 exhibiting a marked increase in signal intensity of the anastomosed vessel (red arrow). **(C and D)** Digital subtraction angiography via left common carotid artery injection **(C:** anteroposterior view, **D:** lateral view) “POD 3”

showing excellent patency of the superficial temporal artery—occipital artery (OA)—anterior cerebral artery (ACA) bypass with antegrade flow through the left STA (black arrow)—OA (white arrow) graft to the territory of the left ACA. The anastomotic sites are patent, yet perfusion to the entire territory of the left A2 was not clearly demonstrated (blue arrow) due to local hemodynamic disturbance.

type might be referred to as a “risky vascular orientation” (see **Figure 7A**). This risky vascular orientation, defined as the microsurgical anatomic angles among the recipient artery (Ra), parent

artery, and branching point of the target artery that makes postoperative hemodynamic changes, including reversal of flow with subsequent watershed shift, is more likely to occur. Here, reversal of

flow was occurred as the correlation between intraoperative findings and retrograde analysis of neuroimaging disclosed that the angle between the Ra and parent artery was almost straight

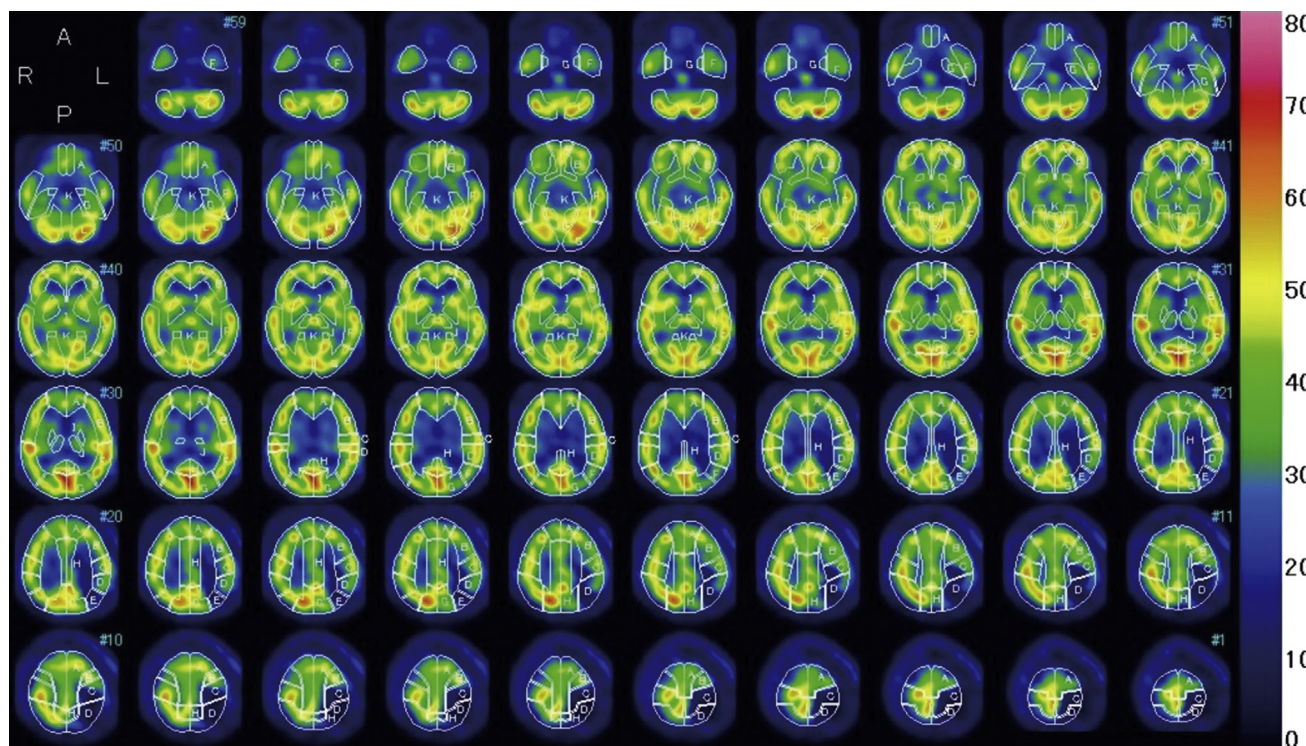
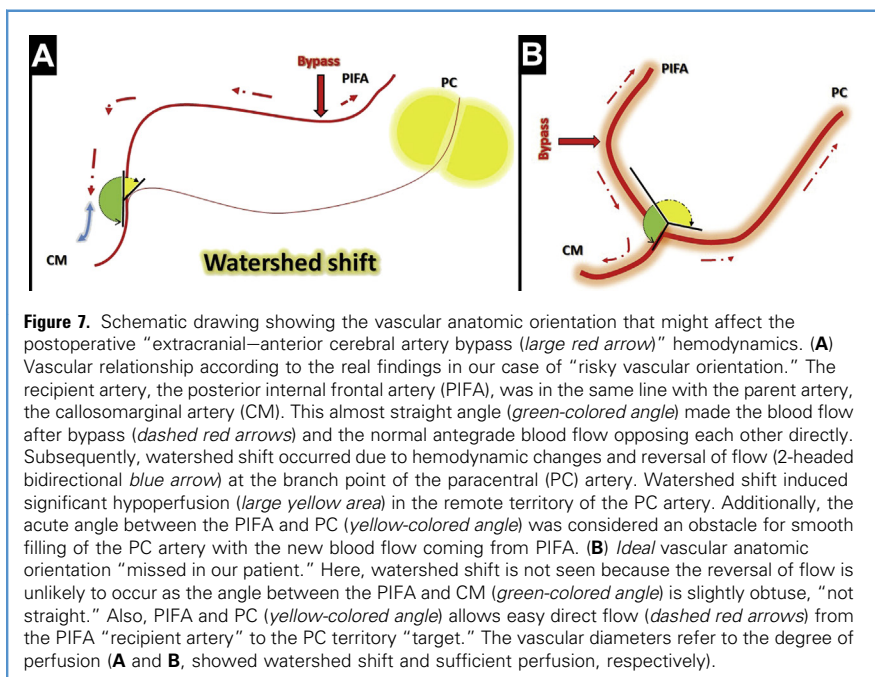
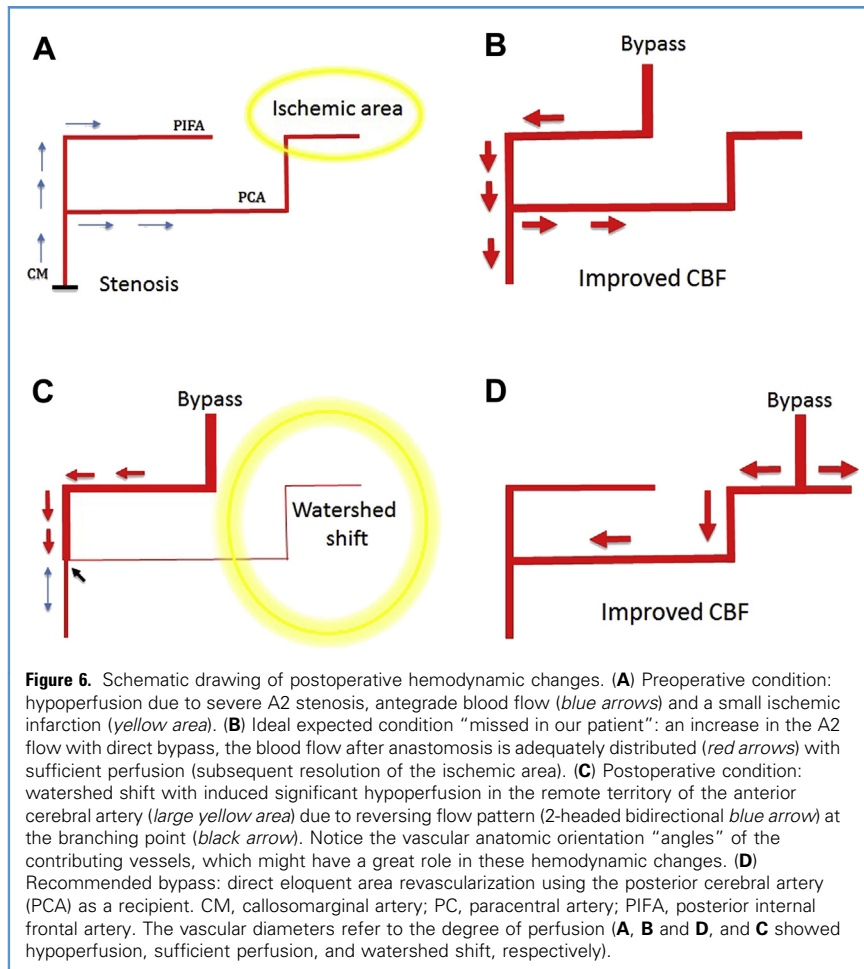


Figure 5. Postoperative resting ^{99m}Tc depreotide single-photon emission computed tomography study obtained 3 days after surgery, revealing improvement of the left cortical uptake. However, the higher portions of

regions (C, D, and E) became markedly hypoperfused (marked decrease in uptake). Watershed shift was confirmed. Colors represent perfusion levels according to the color scale on the right.



(which made “the blood flow after bypass” and “the normal antegrade blood flow” facing each other directly). Additionally, the acute angle between the Ra and target artery was considered an obstacle for smooth filling following bypass. On the other hand, if the angles between these arteries were ideal (Figure 7B), watershed shift did not likely occur.

Better ACA Revascularization Options. Procedures like extensive double bypass can reduce the risk of watershed shift; however, it is not applicable during ACA revascularization. Although IC-IC bypass can provide adequate distal perfusion, it might add a new risk to a high-risk patient. Another option is direct eloquent area revascularization, using the paracentral artery as a recipient (see Figure 6D). This last option might carry a risk for postoperative neurologic deficits due to direct temporary occlusion of the feeding artery during anastomosis. However, in highly skilled hands, this postoperative neurologic deficit might be transient.

Advantages and Limitations of Our Study. On the basis of this study, ACA revascularization in a high-risk patient is not a completely safe procedure. A risky vascular anatomic orientation might be involved in the postoperative hemodynamics and direct the patient outcome. Accordingly, direct eloquent area revascularization might be a better option to prevent significant permanent postoperative neurologic deficits.

The so-called “risky vascular anatomic orientation” and its effect on the postoperative hemodynamic need further studies incorporating a larger number of patients for more solid conclusions. Also, the efficacy of direct eloquent area revascularization in such cases needs to be proved as a better option. Despite we consider these 2 points as limitations of our study, it carries new questions that might be solved in the near future.

CONCLUSION

Our study disclosed that ACA revascularization in a high-risk patient is not a completely safe procedure as it might cause hemodynamic changes and permanent postoperative neurologic deficits. A risky vascular anatomic orientation might

play an important role in the postoperative hemodynamics; accordingly, the surgical plan should be modified.

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