



## Perioperative Bioccipital Watershed Strokes in Bilateral Fetal Posterior Cerebral Arteries During Spinal Surgery

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### Key words

- Cerebral watershed infarction
- Circle of Willis
- Intraoperative hypotension
- Perioperative stroke
- Posterior cerebral artery
- Spinal surgery
- Vision loss

### Abbreviations and Acronyms

BP: Blood pressure  
 CBF: Cerebral blood flow  
 ECG: Electrocardiogram  
 fPCA: Fetal posterior cerebral artery  
 ICA: Internal carotid artery  
 PCA: Posterior cerebral artery

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### INTRODUCTION

Perioperative stroke is a rare complication of general surgery with an estimated incidence between 0.02% and 1%.<sup>1-3</sup> Intraoperative hypotension is considered to be at least a promoting factor, but mechanisms affecting cerebral circulation remain unclear.<sup>4,5</sup> More, it has been suggested that congenital variation of the circle of Willis could potentiate the risk of watershed strokes during intraoperative hypotension.<sup>4,6</sup> Patients undergoing spinal surgery are particularly exposed to intraoperative hypotension with a risk of vision loss due to ophthalmic and cortical infarctions. Watershed ischemic strokes in the posterior cerebral artery (PCA) territories have never been described before. It would support the hypothesis of cerebral hemodynamic insufficiency by decreased

■ **BACKGROUND:** Vision loss due to cerebral infarction during spinal surgery is less described. Intraoperative hypotension would be a leading cause. Patients with variation of the circle of Willis could be more prone to present stroke in this context, but reports are lacking to sustain the theory. Bilateral occipital watershed ischemic strokes have never been described before. We report the case of a patient with a fetal origin of both posterior cerebral arteries (PCAs), presenting this particular anatomic stroke following lumbar laminectomy surgery for spinal stenosis during which intraoperative hypotension was observed. We discuss how this common anomaly associated with intraoperative hypotension could have promoted this serious complication.

■ **CASE DESCRIPTION:** A 55-year-old man woke up with cortical blindness after he had undergone lumbar surgery during which a marked decrease in blood pressure had occurred. Magnetic resonance imaging revealed bilateral symmetric infarctions of the occipital lobes in the distal territory of both PCAs and smaller anterior watershed ischemic strokes, suggesting a hemodynamic mechanism. Extended investigations, including conventional angiography, failed to find any cause of stroke but revealed bilateral fetal PCAs supplied by internal carotid arteries only. Two years later, the patient has not recovered and remains severely visually impaired.

■ **CONCLUSIONS:** The standing hypothesis would be posterior low-flow infarctions resulting from intraoperative hypotension on a variation of the circle of Willis more prone to decrease in cerebral blood flow. Moreover, this case supports the hypothesis of vascular insufficiency due to intraoperative hypotension as cause of stroke during spinal surgery.

cerebral blood flow (CBF) as a mechanism of ischemia.<sup>7</sup> We report a case of a patient with bilateral fetal PCAs (fPCAs) who presented this particular anatomic infarction in the course of spinal surgery during which intraoperative hypotension was applied.

### CASE

The patient gave written consent for the description of the case. The case was reported to the pharmacovigilance center.

A 55-year-old man, without any specific medical history or under any treatment (165 cm, 65 kg, body mass index of 23.9 kg/m<sup>2</sup>), was admitted for scheduled lumbar laminectomy surgery for spinal stenosis. Blood pressure (BP) before surgery was 176/114 mm

Hg. After premedication with 100 mg hydroxyzine orally, anesthesia was induced with inspired sevoflurane 5%, sufentanil 75 µg, and propofol 120 mg. It was maintained with inspired sevoflurane 1% to 3% with an additional dose of 75 µg sufentanil. After induction, the patient was placed in the genupectoral position for surgery. Electrocardiogram (ECG), blood oxygen saturation, capnia, and temperature were monitored throughout the anesthesia, as well as BP with a noninvasive cuff on the right arm. Systolic BP was between 61 and 145 mm Hg with the exception of a 12-minute period at 48 mm Hg. It was lowered to <80 mm Hg for 75 minutes to prevent bleeding. Diastolic BP averaged 53 mm Hg (Figure 1). Total anesthesia time was 95 minutes. No blood transfusion was required. Once ventilation

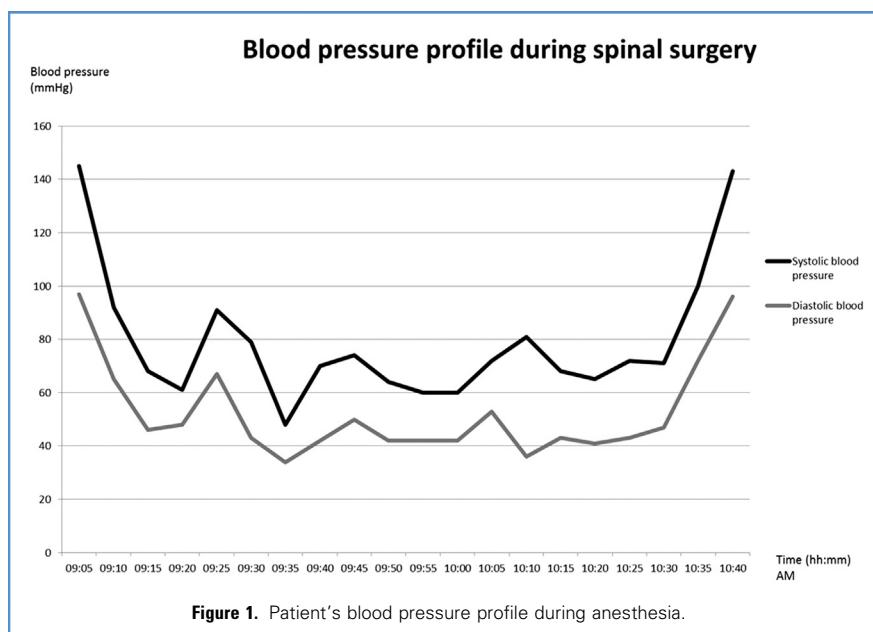


Figure 1. Patient's blood pressure profile during anesthesia.

resumed spontaneously, the patient's trachea was extubated and the patient was taken to the recovery area without any reported incident or cardiac arrhythmia. When he woke up, he complained of complete blindness. Ocular appearance was normal at funduscopic examination with complete pupillary responsiveness. No confusion or memory disturbance was noticed. Brain magnetic resonance imaging revealed extended symmetric areas of recent ischemia in both occipital lobes on diffusion-weighted sequences

(Figure 2A) and a typical aspect of bilateral anterior watershed strokes at the junction of the anterior and middle cerebral arteries (Figure 2B). Cerebral infarctions were already visible as hyperintensities on fluid attenuated inversion recovery sequences. Time-of-flight magnetic resonance angiography did not show any vascular occlusion but revealed a bilateral fPCA with hypoplasia of proximal segments P1 (Figure 3A and B). Blood tests showed a hemoglobin level of 14 g/dL, platelet count of 200 G/L, C-reactive protein level of 9 mg/L, low-density

lipoprotein cholesterol level of 3.16 mmol/L, and preprandial blood glucose level of 6 mmol/L. Coagulation tests were normal. ECG, 48-hour cardiac monitoring, transesophageal echocardiography, and examination of cervical and cerebral arteries by ultrasound were normal. BP monitoring in the stroke unit did not reveal arterial hypertension. Faced with this unusual aspect of ischemia in both PCA territories, a conventional angiography was performed. It showed normal arterial caliber but confirmed the variation of the circle of Willis with both PCAs originating from the carotids through posterior communicating arteries, hypoplastic P1 segment, and distal part of the basilar artery (Figure 3C and D). Unfortunately, the patient's clinical status did not significantly improve during hospitalization. The patient was discharged with paramedical assistance and a daily dose of aspirin. Severe visual impairment and left spatial neglect remain after 2 years.

## DISCUSSION

Vision loss is a complication encountered in 0.2% of spinal surgeries.<sup>8,9</sup> Most cases result from ischemia of the optic nerve or the retina promoted by anemia, intraoperative hypotension, and elevated intraocular pressure due to prone position during a long time.<sup>10</sup> Cortical blindness and cerebral infarction at large are less described.<sup>11</sup> Our case suggests intraoperative hypotension would be a leading cause of stroke in this context. Intraoperative hypotension is widely observed during major spinal surgery due to disruption of extensive vascular networks found in bones.<sup>12</sup> More, it can be applied by physicians to decrease blood loss and avoid transfusion.<sup>13,14</sup> The POISE trial showed that an episode of hypotension at any time during a patient's surgical procedure is related to a higher risk of perioperative ischemic stroke.<sup>5</sup> However, while vascular insufficiency resulting from decreased CBF would seem to be the most logical explanation, no solid evidence supports this theory. There is a discrepancy between the high frequency of intraoperative hypotension and the rare reports of stroke occurring during general surgery, particularly during the intraoperative phase, suggesting that another mechanism is involved. Cerebral

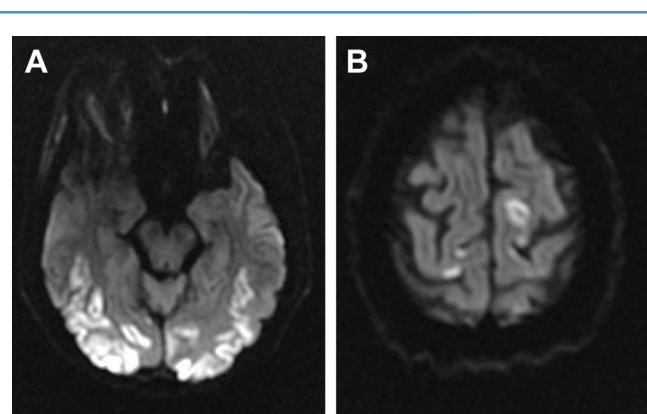
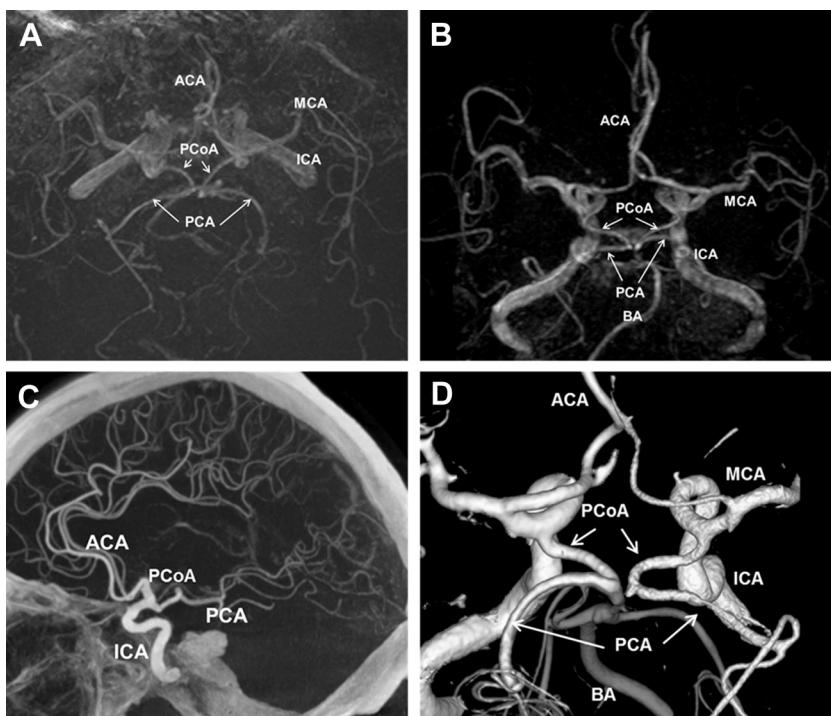


Figure 2. Axial diffusion weighted cerebral resonance magnetic imaging showing recent ischemia in both distal territories of cerebral posterior arteries (A) and recent bilateral anterior watershed ischemic strokes (B).

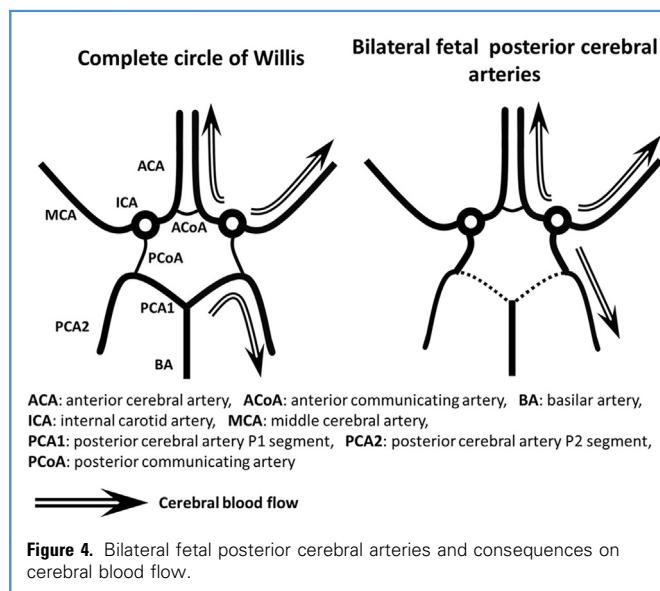


**Figure 3.** Circle of Willis with bilateral fetal posterior cerebral arteries, axial (A) and coronal (B) planes of time-of-flight magnetic resonance angiography, sagittal view (C) of conventional angiography and upper oblique view (D) on 3-dimensional reconstruction. ACA, anterior cerebral artery; BA, basilar artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PCoA, posterior communicating artery.

watershed infarctions as a witness of vascular insufficiency are less described. More cases of cortical infarctions suggesting an embolic mechanism are reported, as perioperative hypotension can cause paroxysmal atrial fibrillation and arterial micro emboli.<sup>15,16</sup> Nevertheless, the aspect of posterior infarction in our case was not typical of stroke due to PCA occlusion. Rather, it seems to be the result of a transitory decreased CBF at the distal parts of the PCAs. Among the arguments supporting this hypothesis of cerebral vascular insufficiency during surgery is the bilateral and symmetric aspect of the posterior infarctions coupled with the presence of typical anterior watershed strokes. Furthermore, thorough investigations failed to find any other cause of ischemic stroke. The addition of two circumstances could explain watershed infarctions of the PCAs in this case. First is the dramatic decrease in BP during spinal surgery, all the more so given the high initial BP values. However,

we failed to identify hypertension in medical history and during careful monitoring in the stroke unit. The

intraoperative BP profile of the patient accords with criteria of definition of intraoperative hypotension given by Bijker et al.: “1) a 20% decrease in systolic pressure from the baseline value 2) a combination of systolic pressures below 100 mm Hg or a greater than 30% decrease from the baseline value, and 3) a systolic pressure below 80 mm Hg.”<sup>17</sup> Prolonged time of intraoperative hypotension would also be determinant.<sup>7</sup> It could result in a decrease of CBF, for which distal cerebral arterial territories are the most vulnerable. Furthermore, we hypothesize that the fPCAs, presented by this patient, made the posterior territory more sensitive to a decrease in CBF. This congenital variation of the circle of Willis is relatively common: the unilateral form is found in 11 to 29% of the population, and the bilateral form in 1 to 9% and is more often found in brains with signs of ischemic infarction on autopsy.<sup>18,19</sup> It is an embryonic derivation of the PCA from the internal carotid artery (ICA) through a patent posterior communicant artery. Consequently, the PCAs depend completely on the anterior circulation and the ICAs have to supply a larger flow territory (Figure 4). Moreover, this disposition and the absence of communication of the PCAs with the vertebrobasilar system would mean that the occipital lobe is a more distal vascular area, more prone to suffering from a



**Figure 4.** Bilateral fetal posterior cerebral arteries and consequences on cerebral blood flow.

decrease in CBF. The presence of typical anterior watershed strokes suggests this variation made all the ICA territory more vulnerable to intraoperative hypotension, in contrast to posterior territories like the cerebellum and brainstem, where no infarction occurred. Drummond et al. have already described a left anterior watershed cerebral infarction following intraoperative hypotension and suggested that a congenital variation of the circle of Willis would potentiate the hemodynamic effect.<sup>6</sup> But this case differs from ours as it concerns a unilateral anterior infarction with an isolated left carotid (i.e., supplying the left anterior circulation only without any communication with the contralateral and posterior circulations). However, both cases indicate that an incomplete circle of Willis increases vulnerability to BP decrease and the severity of the resulting ischemic strokes. Both patients presented large cerebral infarctions, bilateral in our case, and resulted in severe disability. The severity of the strokes could in part be explained by the fact that the central nervous system suffering from ischemia would be less able to regulate BP and respond to drugs correcting intraoperative hypotension, one problem making the other worse.<sup>20</sup> Bilateral infarctions in the PCA territories previously described in the literature differ from our case in several points. These strokes usually result from embolic mechanisms due to atrial fibrillation or artery-to-artery embolization.<sup>21</sup> The latter may even originate from ulcerated atherosclerotic plaques in ICA through persistent cervical embryologic connections with posterior circulation, like hypogloss and trigeminal arteries.<sup>22,23</sup> Fortunately, our patient did not present the classical Dide-Botcazo syndrome, described in bioccipital infarctions, associating anosognosia, amnesia, and topographical disorientation in addition to cortical blindness. This would be explained by the conservation of the inferomedial temporal lobe structures and thalamus usually involved following the complete occlusion of PCAs.<sup>24</sup> We can conclude that a hemodynamic mechanism in our case led to a posterior cortical infarction with a particular location and clinical presentation.

## CONCLUSIONS

The atypical posterior watershed strokes presented by our patient could be the result of a decrease in arterial BP during spinal surgery occurring in a setting of bilateral PCAs. This case supports the hemodynamic insufficiency hypothesis as a mechanism of stroke due to intraoperative hypotension during spinal surgery. It would also suggest that patients with this common variation of the circle of Willis are at higher risk of intraoperative stroke and intraoperative hypotension should be avoided in this context.

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