# Successful aortic fenestration to treat prolonged motor paralysis of the lower extremities after repair of type A aortic dissection

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Paraplegia after repair of a type A aortic dissection is rare. We report a case, which resolved after fenestration was added to lumbar cerebrospinal fluid drainage.

An extra illustration is available online.

## Clinical Summary

A 44-year-old man was seen with chest pain. His troponin level was 4.4 $\mu$g/dL. His pain persisted, and a computed tomographic scan revealed a Stanford type A aortic dissection. Echocardiography showed preserved aortic valve anatomy without pericardial effusion. Magnetic resonance imaging (MRI) showed the origin of the dissection to be distal to the left subclavian artery, with antegrade extension to the right common iliac artery and a retrograde extension along the greater aortic curve to the right coronary artery without evidence of reentry into the aortic lumen proximally (Figure E1).

The patient underwent surgery, where findings confirmed preoperative observations. Cardiopulmonary bypass was carried out through the right axillary artery and the right atrium. The right coronary artery was occluded proximal to the crux and bypassed at the level of the posterior descending artery. The ascending aorta was replaced with a 24 Hemashield graft (Boston Scientific Corporation, Natick, Mass) after stabilization of the false lumen with BioGlue (CryoLife Inc, Kennesaw, Ga). The aorta remained clamped throughout the procedure.

On awakening, the patient was found to have complete motor paralysis of his lower extremities but with preserved sensation. Methylprednisolone was administered at a dose of 7 mg/kg. Lumbar drainage was instituted with a Codman catheter (Codman and Shurtleff, Inc, Raynham, Mass), adjusted to maintain a cerebrospinal fluid pressure of 7 cm $H_2O$. The patient was weaned from mechanical ventilation. Motor paralysis persisted.

A fenestration procedure was initiated 24 hours postoperatively, 16 hours after the paralysis was first noticed. With the patient under conscious sedation, pigtail catheters were positioned into both the true and false lumina of the thoracic aorta through the femoral arteries. Images revealed flow in the false lumen of the thoracic aorta to be stagnant. Multiple intercostal arteries were seen arising from the false lumen. No thrombus was seen. Aortography through the dominant true lumen failed to opacify the false lumen in the chest (Figure 1, *A* and *B*). There was communication between the two lumina in the abdomen, with antegrade flow in the false lumen in the abdomen. A Colapinto catheter (Cook, Inc, Bloomington, Ind) was placed into the true lumen, and a Rosch-Uchida needle (Cook) was advanced through the pigtail catheter in the false lumen at the midthoracic level. Aortic fenestration was achieved with a 14-mm balloon. This was confirmed by the observation of brisk flow from the true lumen to the false lumen of the descending thoracic aorta, where flow had previously been noted to be stagnant. The pressure difference between the two residual lumina was minimal. (Figure 1, *C*)

The patient had immediate increased mobility of his feet. After 48 hours, the patient was able to abduct and adduct at the hip, and after 72 hours, he was able to flex at the hip and extend at the knee. The lumbar spinal drain was discontinued after 72 hours. On postoperative day 13, the patient walked with assistance. He continued to have episodic bladder and bowel incontinence. MRI on day 26 showed no evidence of spinal cord infarction.

## Discussion

This report illustrates an unusual case of retrograde type A aortic dissection.[1] The recommended treatment of delayed paralysis after surgery for aortic dissection is lumbar cerebrospinal fluid drainage and high-dose intravenous steroids.[2] Such a treatment was insufficient in our case.

Fenestration to relieve peripheral ischemia caused by dissection has proved effective.[3] The decision to perform fenestration requires analysis. Type A dissection is considered an emergency; however, organ or spinal cord malperfusion might prioritize reperfusion relative to surgical repair.[4]

At some point during or after surgery, our patient's spinal circulation became compromised. The findings at aortography were notable in that both lumina communicated in the abdomen. There was stagnation of blood in the false lumen, which gave off numerous intercostal vessels. Presumably, cardiopulmonary bypass aggravated a tenuous cord perfusion.

Late MRI did not show evidence of spinal cord infarction, suggesting that ischemia was present for nearly 24 hours. This supports the hypothesis that paraplegia after aortic surgery may reflect ischemia rather than infarction. MRI with weighted imaging

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Received for publication Feb 1, 2005; accepted for publication Feb 8, 2005.

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J Thorac Cardiovasc Surg 2005;130:599-600

0022-5223/$30.00



doi:10.1016/j.jtcvs.2005.02.009

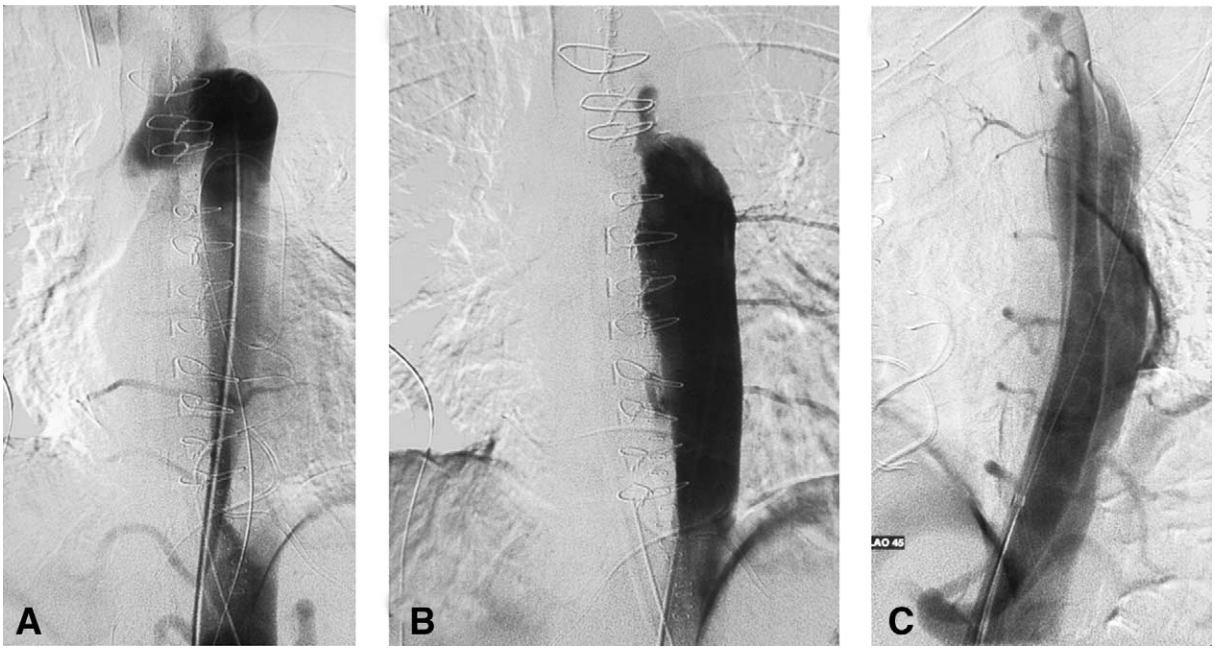


**Figure 1. Digital subtraction aortogram showing contrast opacification of true lumen (A) and false lumen (B). C, Postfenestration aortogram showing contrast opacification of both true and false lumina.**

may provide a tool to differentiate these causes.[5] Such studies, however, are not always feasible early after surgery.

To our knowledge, this is the first report of fenestration to treat spinal cord ischemia complicating aortic dissection. The evolution of this case suggests that cord ischemia may be prolonged without infarction.

## References

1. Von Segesser L, Killer I, Ziswiler M, Linka A, Ritter M, Jenni R, et al. Dissection of the descending thoracic aorta extending into the ascending aorta. *J Thorac Cardiovasc Surg.* 1994;108:755-61.
2. Cheung AT, Weiss SJ, McGarvey ML, Stecker MM, Hogan MS, Escherich A, et al. Interventions for reversing delayed-onset postoperative paraplegia after thoracic aortic reconstruction. *Ann Thorac Surg.* 2002;74:413-9.
3. Slonim SM, Miller DC, Mitchell RS, Semba CP, Razavi MK, Dake MD. Percutaneous balloon fenestration and stenting for life-threatening ischemic complications in patients with acute aortic dissection. *J Thorac Cardiovasc Surg.* 1999;117:1118-27.
4. Deeb GM, Williams DM, Bolling SF, Quint LE, Monaghan RN, Sievers J, et al. Surgical delay for acute type A dissection with malperfusion. *Ann Thorac Surg.* 1997;64:1669-75.
5. Kuker W, Weller M, Lose U, Krapf H, Dichgans J, Nagele T. Diffusion-weighted MRI of spinal cord infarction—high resolution imaging and time course of diffusion abnormality. *J Neurol.* 2004; 251:818-24.

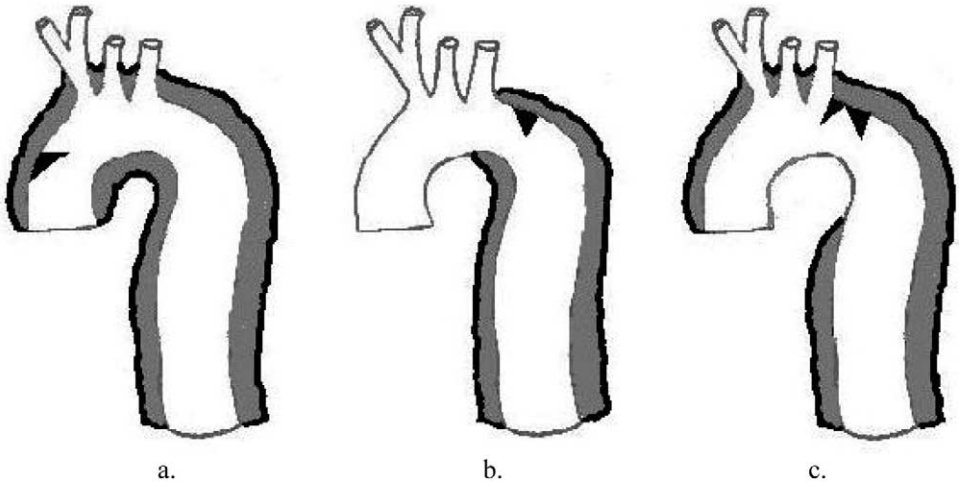


**Figure E1. a, Stanford type A dissection. b, Stanford type B dissection. c, Retrograde dissection more accurately exemplifies this patient's diagnosis and is thus difficult to fit into typical classification.**