Hemodynamics of Aortic Dissection

Chris Elkins and Michael D. Dake

Contents

3.1 Introduction and Background ..................... 27
3.2 Experiments on the Hemodynamics of True-Lumen Collapse ..................... 28
3.3 Causative Factors in True-Lumen Collapse .......... 29
3.4 Effective Treatment for True-Lumen Collapse .......... 30
3.5 Conclusions .................................. 31

3.1 Introduction and Background

Aortic dissection is the most frequent nontraumatic catastrophe that affects the aorta, with an annual incidence exceeding that of spontaneous rupture of aortic aneurysms [1]. Aortic dissection occurs with a frequency of 10–20 cases per million population per year. Approximately 30% (85 of 272 [2], 106 of 325 [3]) of patients with aortic dissection have one or more ischemic complications of the peripheral vasculature, including stroke, paraplegia, loss of peripheral pulses, and compromised renal or mesenteric perfusion. The surgical mortality rates for patients with acute aortic dissection complicated by compromise of a peripheral arterial branch exceed 50% [3]; visceral and renal ischemia are important independent predictors of death as a result of surgery [2].

In the past, the direct propagation of a dissection flap into an aortic branch with the resultant compromise or obstruction of the true lumen was considered to be the basic mechanism for ischemic complications in the peripheral vasculature. This understanding was based on observations of cross-clamped or decompressed aortas without flow and on findings at necropsy.

Recently, collapse or obliteration of the true lumen was proposed as another important mechanism for compromise of the aortic branch in aortic dissection [4, 5]. This is based on antemortem cross-sectional imaging studies, including those performed with intravascular ultrasonography, that facilitate an appreciation of the effects of flow on the anatomic relationships between the flap, aortic lumina, and branch vessels [4, 5]. In this setting, the plane of the dissection flap spares the branch vessel. Instead, the flap is positioned in a curtainlike fashion across the origin of the vessel, which causes dynamic obstruction of the branch artery [5]. According to the report by Williams et al. [6], dynamic obstruction due to true-lumen collapse was the cause of the infradiaphragmatic organ or limb ischemia in 20 of 24 patients. Among the 20 patients, 14 had ischemia in multiple organs that involved the mesenteric, renal, and lower-limb circulations. Recently, percutaneous endovascular treatment with balloon fenestration and stent placement was introduced to relieve true-lumen collapse and showed promising results [1, 5, 6]. However, there have been few clinical and experimental studies conducted to investigate the causes of true-lumen collapse in aortic dissection and the possible treatment methods to relieve true-lumen collapse or to determine the most effective methods.

Patients with chronic dissection often develop late complications mainly related to the patency of the false lumen [7]. In these cases, there is progressive dilatation of the false lumen that can lead to eventual rupture. An acute aortic diameter of greater than 4 cm in type B dissections has been found to be an indicator of possible future rupture [8, 9]. While some studies suggest medically treating dissections with maximum diameters less than 5 cm [10], others support surgery or placement of stent-grafts when the false lumen is greater than 4 cm [8], 5 cm [11], or 6 cm [12] in order to avoid certain rupture in the future. Obviously, there is considerable uncertainty about the critical diameter of the false lumen and how to treat false lumen aneurysms.

While formation and development of dissections are not well understood, it is generally accepted that hemodynamics plays a major role in the initiation, acute propagation, and chronic development of dissections. In all three of these stages, hemodynamic effects couple with mechanical and biological processes in the arterial walls. It may be some time before the initiation and propagation mechanics of dissection will be understood.
as they involve hemodynamic forces interacting with the aortic wall in both its healthy and its diseased state. There are many studies of the hemodynamic effects in aneurysmal growth. In most cases, aortic dilatation evolves slowly over several years (1 mm per year [10]) and can be treated medically. Relatively less effort is being spent on understanding the problem of branch vessel ischemia and, in particular, the special case of true-lumen collapse. Yet, this problem is of critical importance in both acute and chronic dissection and is highly related to hemodynamics.

### 3.2 Experiments on the Hemodynamics of True-Lumen Collapse

An in vitro study at Stanford created two aortic dissection phantoms to investigate the causative factors for true-lumen collapse and to develop effective treatments [13, 14]. One phantom was compliant and opaque (Fig. 3.1), and the other was rigid and transparent (Fig. 3.2). The rigid, transparent phantom was created to allow visual observation of the true lumen along the length of the aorta. Each phantom had the following physical features to model a Stanford type B aortic dissection: an aortic arch, true and false lumens with abdominal branch vessels, and a distal bifurcation. These phantoms were placed in a pulsatile mock-flow loop, with water as the working fluid. The effects of anatomic factors (entry-tear size, branch-vessel flow distribution, fenestrations, distal reentry communication) and physiologic factors (peripheral resistance in the branch vessels, pump output and rate, vascular compliance) on true-lumen collapse were investigated. The morphology of the true lumen was observed. Branch pressures and flow rates were measured.

After true-lumen collapse had been induced, experiments were conducted to evaluate the effectiveness of clinically relevant variables in relieving the collapse. Variables included entry-tear size, branch-vessel flow distribution, distal reentry communication between the true and false limbs, aortic fenestrations, and pump output. To test the effect of closing the entry tear, a

![Fig. 3.1](image1.png) Compliant model of aortic dissection. Compliant and opaque phantom model of type B aortic dissection with entry tear, true and false lumens, branch vessels, and distal bifurcation. This phantom was placed in a pulsatile mock-flow loop to observe and measure the effects of a variety of anatomical and physiological factors on branch-vessel flow rates

![Fig. 3.2](image2.png) Rigid model of aortic dissection. Rigid and transparent model of type B aortic dissection allowed visual observation of the true lumen along the length of the aorta. This allowed direct evaluation of the relative effectiveness of altering clinically relevant variables in relieving the complications of dissection with aortic true-lumen collapse