8 Presentation in a Shocked State: The Impact and Management of Pericardial Tamponade

Christian Olsson

Abstract

In acute type A aortic dissection, tamponade is caused by intrapericardial aortic wall oozing, contained or free aortic rupture. It can develop insidiously or abruptly and if not self-limiting will result in circulatory collapse and shock. Often occurring in more than a third of surgical patients, tamponade is one of the most common severe complications of acute type A aortic dissection, also identified as the most common cause of death in non-operated dissection victims. In clinical series, mortality approaches 50 % and in multivariable statistical analyses of risk factors, tamponade and shock are frequently among the most prominent found. Fluid replacement and other resuscitation measures are necessary for patient stabilization and immediate surgical repair the key to improved outcome. If surgery is not immediately available, a pericardial drain can be placed to evacuate tamponade in the shocked or hemodynamically compromised patient. In the operating theatre, a swift procedure aiming at restoring circulation is paramount, by first relieving the tamponade or by instituting extracorporeal circulation as circumstances dictate.

Keywords

Tamponade • Shock • Pericardial drain • Surgery • Outcomes

Clinical Vignette

A 76-year old man, previously healthy, arrives in the emergency department with chest pain, blurred vision and weakness and discomfort of the right arm. Symptoms abate, ECG and troponins are normal and he is admitted to a neurological ward. Head CT shows no acute insult. In the evening chest pain recurs and troponins are slightly elevated; he is transferred to the CCU. In addition to salicylate, he is administered clopidogrel and fondaparinux pending confirmation of acute coronary syndrome. A murmur is auscultated and the next morning TTE is performed, showing ascending aortic dilatation (58 mm), dissection membrane, moderate-severe (grade III) aortic regurgitation, and pericardial exudate

C. Olsson, MD, PhD
Cardiovascular Surgery Unit, Department of Molecular Medicine and Surgery, Karolinska University Hospital, Karolinska Institutet, Stockholm SE 17176, Sweden
e-mail: christian.olsson@ki.se

R.S. Bonser et al. (eds.), *Controversies in Aortic Dissection and Aneurysmal Disease*,
DOI 10.1007/978-1-4471-5622-2_8, © Springer-Verlag London 2014

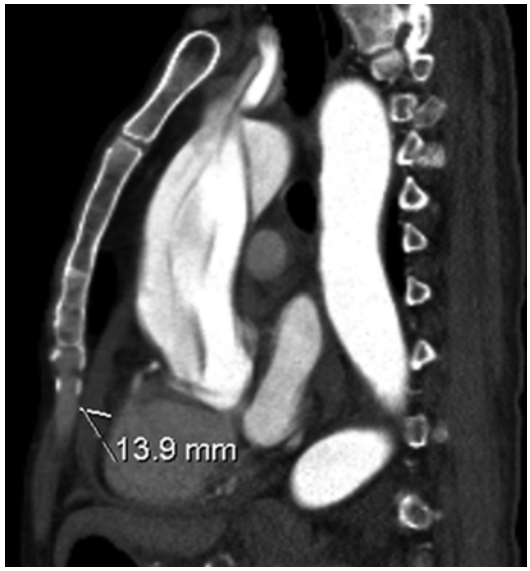

Fig. 8.1 Chest CT (sagittal plane) of patient in case presentation approximately 48 h after presentation showing ascending aortic dissection (DeBakey type II) extending into the brachiocephalic trunk and 14 mm pericardial fluid ventral to right outflow tract

(12–16 mm) without hemodynamic compromise. After consultation, CT of the aorta is performed, finally establishing the diagnosis of DeBakey type II aortic dissection involving the brachiocephalic trunk and, 14 mm pericardial exudate (Fig. 8.1). Thus, 50 h after presentation, the patient is accepted for emergent surgery. Still hemodynamically stable, he is transferred and arrives in the operating theatre 1.5 h later where he suddenly decompensates and collapse. Based on the suspicion of severe tamponade, he undergoes a rapid sternotomy and pericardiotomy to relieve pressure, by which he swiftly exsanguinates from an aortic rupture (subsequently localized as a 2 × 2 cm hole in the posterior wall at the junction of the ascending and arch parts of the aorta). Without bleeding control, further measures are judged futile and the surgical procedure is aborted.

This and similar clinical cases, poses several questions regarding tamponade and associated shock in acute type A aortic dissection.

What Causes Tamponade and Shock in Acute Type A Aortic Dissection?

Shock is the clinical, premortal end-stage of a final common pathway leading to circulatory collapse (Fig. 8.2). Definitions of shock vary, but most would agree that systolic blood pressure <90 mmHg and clinically evident hypoperfusion of at least one end-organ (manifesting as altered consciousness or other non-focal neurological symptoms, oliguria or anuria, hypoxia, arrhythmia, or peripheral vasoconstriction) are the hallmarks. In the setting of acute type A aortic dissection, tamponade is the most frequent cause of shock, when severe causing a very harmful combination of hemorrhagic hypovolemia and forward pump failure by impeded preload. Tamponade, in turn, will almost certainly develop in the presence of free intrapericardial aortic rupture. But tamponade may also develop in a more tempered process resulting either from a contained (covered by delicate adventitial tissue) rupture or from ascending aortic oozing, often with typical surgical findings of a blood-imbibed aortic wall, para-aortic hematoma and sometimes a hematoma spreading to adjacent heart structures—the right ventricular outflow tract, the main pulmonary artery, and the roof of the atria. In fact, pericardial fluid and tamponade seems at least as common with intramural hematoma as with classic dissection: 63 % vs. 37 % and 49 % vs. 31 %, respectively, in recent studies [1, 2]. Hypothetically, this relates to a more superficial aortic wall damage in combination with intramural pressurization in the absence of reentry [2], and may serve as one of several reasons to treat these conditions in a common clinical context. As described in Fig. 8.2, shock can be hypovolemic (hemorrhagic), cardiogenic, metabolic, neurogenic or a combination of these in origin. Exsanguination by bleeding into the mediastinum or pleura will result in hypovolemic shock if not adequately fluid resuscitated. Dissection into either coronary artery can result in myocardial ischemia with pump failure and/or malignant arrhythmia with sometimes immediately life-threatening cardiac collapse.

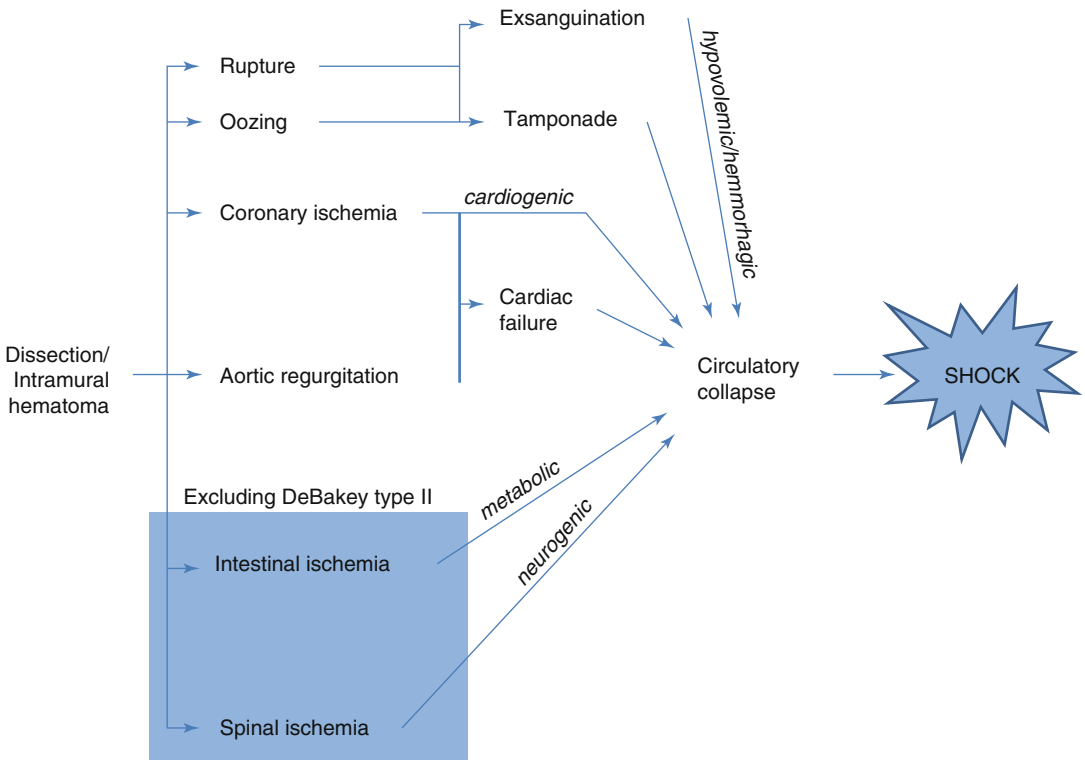

Fig. 8.2 Schematic illustration of acute dissection (or intramural hematoma) complications leading to circulatory collapse, and ultimately shock, with different pathophysiological background. The boxed conditions are possible only with DeBakey type I extent of dissection

Dissection or intramural hematoma extending into the aortic root interfering with valve supporting structures commonly causes varying degrees of aortic regurgitation. When regurgitation is severe and developing rapidly, the heart may decompensate with ensuing pulmonary oedema and shock. In this scenario, beta-blockers, if at all indicated to control blood pressure, must be used cautiously, avoiding increased regurgitant fraction by induced bradycardia. Intestinal ischemia and spinal ischemia may develop with DeBakey type I dissection extending beyond the arch. The deleterious lactacidosis produced by intestinal ischemia can precipitate metabolic derangement and collapse, and the infrequent spinal ischemia, especially when more cephalad and not involving only the anterior cord, can result in so-called spinal shock mainly due to neurogenic vasoplegia with loss of resistance, perfusion pressure and relative hypovolemia from peripheral pooling of blood.

The case presentation illustrates the transition from clinically insignificant pericardial effusion (14 mm on CT) to lethal tamponade and aortic rupture in 1.5 h, emphasizing the tenuous state of the aorta and the tendency for this lethal complication to occur abruptly.

How Common Is Tamponade and Shock in Acute Type A Aortic Dissection? Can it Be Prevented or the Risk Diminished?

In the limited but detailed autopsy-study from Toronto [3], tamponade was the most common cause of death in non-operated patients with acute type A aortic dissection, found in 23/29

Table 8.1 Summary of prevalence of tamponade and shock and their related surgical mortality

Author [Ref no]	Year	n	Shock		Tamponade	Mortality, overall
Ehrlich et al. [4]	1998	109	23 (21 %)[a]		30 %	48 %
Bayegan et al. [5]	2001	87	28 (32 %)	40 (46 %)	30 %[b]	–/30 %
Tan et al. [6]	2001	252	31 (12 %)/58 (23 %)[c]	64 (25 %)	25 %	39 %/31 %
Mehta et al. (IRAD) [7]	2002	547	154 (29 %)[d]		27 %	47 %
Chiappini et al. [8]	2004	487	50 (10 %)	100 (20 %)	22 %	–
Rampoldi et al. (IRAD) [9]	2007	682	148 (12 %)/193 (31 %)[c, d]		24 %	30 %
Santini et al. [10]	2007	311	74 (24 %)	121 (39 %)	23 %	–
Gilon et al. (IRAD) [11]	2009	674	–	126 (19 %)	20 %	–/44 %
Girdauskas et al. [12]	2009	276	53 (19 %)	38 (14 %)	19 %	–
Goda et al. [13]	2010	301	87 (29 %)	83 (28 %)	14 %	18 %/16 %

IRAD International Registry of Acute Aortic Dissection
[a]Defined as hemodynamic instability
[b]Excluding six patients dying preoperatively
[c]At presentation/at beginning of surgery
[d]Defined as hypotension/shock/tamponade

(79 %), with concomitant ascending aortic rupture in five, suggesting that tamponade is common, and lethal if not timely and adequately handled. In clinical series of operated patients (Table 8.1), tamponade is reported in 14–46 % and shock in 10–32 % of patients, respectively [4–13]. This makes tamponade and associated circulatory collapse among the most common severe complications of acute type A aortic dissection; malperfusion syndromes are reported prevalent in 10–25 % (most commonly affecting lower limb or kidney) [10, 12] and aortic regurgitation in 18–70 % [6, 13].

The inciting event for tamponade, the intimal tear, cannot in itself be prevented by other means than those directed at preventing aortic dissection; control of hypertension, surveillance of patients-at-risk, pre-emptive elective aortic repair. The time between the onset of dissection and the development of hemodynamically significant tamponade, however, is a "window of opportunity". As emphasized by numerous authors [7, 8, 10, 13], the rapid identification of tamponade and its definite surgical management is probably a key to avoid the severe consequences of circulatory collapse. Illustratively, in reports from IRAD [7] and the Netherlands [6], some 12 % of patients presenting with tamponade/shock more than double to 23–31 % at the beginning of surgery (Table 8.1), i.e., develop a hemopericardium of clinical significance during diagnostic work-up or other measures delaying operation. Strikingly apparent in the report from Goda et al. [13], tamponade and shock were, uncommonly, in themselves *not* associated with worse outcome, in the authors' interpretation thanks to the expeditious fast-tracking to surgery: 1.6 h from admission to operation in patients with tamponade vs. 4.7 h overall. An applaudable achievement mirrored by an overall low 14 % in-hospital mortality in 301 patients and probably contributing to the elimination of tamponade and shock as independent risk factors.

A clinical problem of continuously changing character is coupled to the misinterpretation of aortic dissection (type A and type B) as acute coronary syndrome or myocardial infarction, occurring in as many as 32 % of cases [14]. A decade ago, this would sometimes prompt inadvertent thrombolytic therapy with irreversible detrimental effects on coagulation by the time of correct diagnosis and emergent surgical treatment. The causative role of thrombolysis in aggravating or eliciting tamponade is not affirmed, but it clearly make circumstances very dire [15, 16] with a reported case fatality rate of 71 % [17]. Today, thrombolysis is replaced by acute percutaneous intervention, by which angiography will often lead

to correct diagnosis in the absence of anticipated coronary pathology. These patients will almost invariably be pretreated with (loading doses of) antithrombotic agents including acetylic acid, clopidogrel, fondaparinux, and heparin; most of them with irreversible effects on platelet function. In the setting of acute dissection, with a primary dysthrombotic state, coupled with extracorporeal circulation and often deep hypothermic circulatory arrest, bleeding diathesis can be overwhelming. In the readable study by Hansen et al. [14], patients with acute aortic dissection who erroneously received fibrinolytic/antiplatelet/antithrombotic therapy had a higher rate of the composite endpoint death or major bleeding (54 % vs. 23 %), a higher frequency of pericardial tamponade (50 % vs. 25 %), and, in surgically treated patients, more reoperations for bleeding (41 % vs. 21 %, not a statistically significant difference).

Even before confirmatory imaging, clues in the presentation may indicate tamponade. Clinically, the so-called Beck's triad of low blood pressure, distended neck veins and distant heart sound suggests tamponade, however with a low negative predictive value. Illustrating valuable information from the medical history, an IRAD study [11] found that syncope, coma, and altered consciousness were three times more common in patients with tamponade (33 vs. 11 %), and in another study tamponade (18 vs. 12 %) and shock (29 vs. 14 %) were more common in patients with malperfusion syndromes [12]. Previous cardiac operation, on the other hand, is often considered protective against tamponade, because of the pericardial adhesions regularly present. In our own study of 360 patients operated for acute type A aortic dissection none of those with tamponade (n = 126) was previously operated [18]. In the study by Gilon et al. [11], 7.0 % of patients with tamponade had previous cardiac surgery, compared to 17 % of those without tamponade ($p = 0.007$), suggesting a decreased risk, but notably, *not ruling out* tamponade even in this setting.

The case presentation represents an awkward reality, *with misinterpretation of symptoms*, *misdiagnosis*, *initiation of ACS regimen and delay of surgery—a combination heralding a disastrous outcome*.

How Should Tamponade (Shock) Be Managed Preoperatively: Before Definite Surgical Repair?

Standard shock and resuscitation algorithms generally apply in aortic dissection, bearing in mind the diversity in aetiology (Fig. 8.2) and the comparably high prevalence of tamponade to direct measures appropriately. Fluid resuscitation and volume expansion remain mainstays of therapy, supplemented with inotropic or vasopressor support to achieve acceptable blood (perfusion) pressure of at least 90 mmHg systolic. Oxygen supplementation, buffering of acidosis and electrolyte correction are also natural parts of initial management aiming at stabilizing the patient until surgery. Formal cardiopulmonary resuscitation including defibrillation, intubation, and mechanical ventilation may also be indicated in critical conditions. It remains sensible to achieve stabilization prior to surgery, if it is not immediately available. However, given the prevalence of tamponade as the underlying cause of circulatory collapse and shock, it is prudent to consider pericardial drainage before surgery. In an earlier Boston report [19], seven normo- or hypotensive patients with acute type A aortic dissection had cardiac tamponade and four underwent pericardiocentesis. Within 40 min, three had sudden onset of electromechanical dissociation and died, whereas the three patients who did not have or underwent unsuccessful pericardiocentesis proceeded to surgery and survived, raising the question—is pericardiocentesis harmful? Tan et al. [6], on the other hand, found that preoperative pericardial drainage of tamponade was associated with *decreased* risk of in-hospital mortality in surgery for acute type A aortic dissection (18 vs. 25 %), stating that hemodynamic compromise resulting from tamponade should be treated promptly, by immediate establishment of extracorporeal circulation or, if not feasible, by pericardial drainage. Svensson [20], in a commentary on Van Arsdell's autopsy study [3], concur: "Drainage of the pericardial fluid to resuscitate a patient is an important method to prevent death before surgery, even though this may precipitate free rupture". A difficult topic to study—and to

settle upon—it would seem that (1) pericardial drainage should be in the form of an indwelling catheter, making possible controlled (ie. interrupted or interval) fluid drainage; (2) pericardial drainage is not indicated, even with imaging evidence of hemopericardium, in a clinically stable patient planned for immediate surgery; (3) pericardial drainage can be life-saving and is an appropriate measure to resuscitate a patient in shock. Facing a lengthier (more than approximately 2 h) transportation to a referral centre, the decision becomes even harder: prophylactically place a drain in an attempt to secure the transportation or "wait and see"? In the absence of evidence, a suggestion would be to place a drain in the patient with a sizeable pericardial effusion (more than 15–20 mm on TTE or CT), draining actively only if circulation deteriorates during transportation. If clot constitutes the hemopericardium, as is sometimes the case, drainage is futile and the haemostatic effect of the clot should be left undisturbed.

In an intriguing trial by Noera et al. [21], patients arriving in the emergency department in shock were immediately (20–40 min after the emergency call) randomized to treatment with standard resuscitation (fluid, inotropic support) or standard therapy supplemented with an intravenous bolus of a synthetic ACTH-analogue. In patients with acute type A aortic dissection with tamponade and shock (n = 32), those treated with the ACTH-analogue arrived in the OR (3–5 h later) with a significantly better blood pressure and also had a much better survival, 13/15 vs. 7/18 in the control group (p = 0.02). Unfortunately, these favourable results have not to date been repeated, much less so in a larger randomized trial. In such a trial, the timely administration of the ACTH-analogue will be critical, and the conceived value of the treatment would further increase could it be shown that ACTH-analogue given at a later stage, after definitive diagnosis or even in the OR at anaesthetic induction, would be equally beneficial.

In the presented case, the literally instantaneous rupture and tamponade left no room for other than very immediate surgical measures. The literature would not support the placement of a pericardial drain in a circulatory stable patient with 14 mm pericardial fluid scheduled for acute operation at arrival after a short (30 min) transportation by ambulance.

How Should Tamponade Be Managed Intraoperatively: To Avoid Disaster?

The patient arriving in the operating theatre with severe tamponade and shock represents a formidable challenge for the surgical team and the importance of leadership, communication, teamwork and skill to control the situation become evident. Any patient surviving this far deserves the chance of surgical repair. On the other hand, heroic attempts to revert the irreversible may be futile at best, or even unethical.

Self-evidently, in the crashing patient, continuous CPR, ventilation, pharmacological support and gentle anaesthesia are necessary. If marginal but stable, peripheral cannulation under local anaesthesia in the awake patient can be considered, to avoid hypotension at induction eliciting a vicious circle. With ongoing rescuing manoeuvres, draping, or even washing and monitoring including venous or arterial lines, can be postponed not to delay the procedure. Heparin should be administered immediately, if need be in a supranormal dose to allow institution of extracorporeal circulation as soon as possible and without necessarily controlling ACT level. At this point the decision must be made to either first evacuate the tamponade surgically by a subxiphoidal incision or by the full sternotomy and a limited pericardiotomy, or to re-establish perfusion by extracorporeal circulation. The latter alternative will almost always entail femoral artery cannulation. If the extent of dissection is favourable for femoral artery cannulation this is the natural step. However, if axillary artery cannulation was planned or considered preferable, this would probably be too time-consuming, favouring initial decompression of the tamponade. In any hands, this will be the quickest approach, also recognizing that femoral artery cannulation can be challenging, due to the dissection and especially in shock, with absent pulses, vasoconstriction

and severe hypovolemia. Similarly, in the very obese, in patients with peripheral vascular disease or in reoperations, it may prove prudent to first relieve tamponade, then proceed with cannulation, with the added luxury of time provided by a stabilized circulation. Failure to relieve tamponade and restore perfusion can cause irreversible cardiac and cerebral damage. Overzealous volume replacement and vasopressor support can be counterproductive at this stage, often entailing a marked blood pressure overshoot when the tamponade is cleared and circulation recovers, increasing the risk of overt aortic rupture and even venous reservoir flooding. If evacuating the tamponade is insufficient in restoring circulation, the procedure must proceed swiftly, assuming additional complications (e.g., myocardial ischemia, intestinal ischemia or aortic regurgitation) as indicated by preoperative studies, and establishment of extracorporeal circulation must not be delayed; after a sternotomy also allowing unorthodox approaches as direct aortic cannulation [22] or cannulation through the left ventricular apex. Unloading the heart will relieve myocardial ischemia. With severe aortic regurgitation, venting of the left ventricle will be immediately necessary to avoid distension. If intestinal ischemia is suspected or verified, every effort must be made to restore flow in the lumen supplying (in order of importance) the superior mesenteric artery, the celiac trunk, and the inferior mesenteric artery which would normally be the true lumen; hence the rationale not to abandon a carefully planned axillary artery cannulation strategy in favour of femoral artery cannulation. The remainder of the procedure is carried out as planned. If no frank rupture is found and resected, the tamponade is reasonably the result of oozing, indicating an especially fragile aortic wall and an incentive not to leave a cross-clamped part of the aortic wall behind.

The patient in the case presentation underwent a sternotomy and pericardiotomy and immediately exsanguinated. In retrospect, a subxiphoidal incision or a very small opening of the pericardium could have sufficed to drain 1–200 ml of blood and to restore acceptable circulation and allow for establishment of extracorporeal circulation to control the situation.

What Influence do Tamponade and Shock Have on Surgical Outcomes?

A very common notion in the literature on risk factors for death in acute type A aortic dissection is that patient- and dissection-related factors takes preponderance over surgical and other treatment (i.e., modifiable) factors [23, 24]. Tamponade or shock will almost invariably surface as prominent risk factors for surgical (30 days, in-hospital) mortality in acute type A aortic dissection, with case fatality rates approaching 50 % [4]. Hypotension/shock/tamponade showed an odds ratio of 3.0—3.2 in two separate IRAD studies [7, 9]. Odds ratio for death was 7.4 for hypotension in the report from Santini et al. [10] and 16 for severe (pulseless) tamponade in another study [5], analyzing a composite endpoint of death or multiorgan failure. The studies by Chiappini et al. [8] and Goda et al. [13], respectively, deviate somewhat from this pattern by reporting not tamponade or shock per se as risk factors, but identifying its most ominous clinical appearance, cardiopulmonary resuscitation, as statistically significant predictor of in-hospital mortality, with odds ratios of 2.2 and 4.0, respectively. In the former study, preoperative tamponade as such was also associated with re-exploration for bleeding (relative risk 3.9).

The Penn Classification: Clarifying the Impact of Generalized Ischemia

In short, suggested risk models [6, 7, 9, 10] for acute type A aortic dissection have failed to be universally adopted. Summarized in Table 8.2,

Table 8.2 The penn classification of acute type a aortic dissection

Penn class Aa	No ischemia
Penn class Ab	Localized ischemia[a]
Penn class Ac	Generalized ischemia
Penn class Abc	Localized and generalized ischemia

[a]With the exception of coronary ischemia, because the clinical ischemic manifestation is that of a circulatory collapse with generalized ischemia

the Penn classification [25], so named after its conception at the University of Pennsylvania, Philadelphia, the repeated and coherent findings of studies on preoperative risk factors for in-hospital mortality in acute aortic type A dissection are elegantly and intuitively grouped by the truly important correlate of malperfusion syndromes and circulatory collapse alike, namely ischemia. According to the Penn classification, ischemia is either localized, i e., engaging one or more end-organs, or generalized, i e., caused by circulatory collapse, or a combination of both. The Penn classification collects the variety of malperfusion syndromes (cerebral, limb, renal, spinal, intestinal) into one category and the variety of critical preoperative states (hypotension, shock, tamponade, congestive heart failure, cardiopulmonary resuscitation, and so forth) into one category, pivoting on clinical ischemia. Hence, e.g., radiological signs of branch vessel occlusion without a clinical correlate are not equivalent to ischemia, and echocardiographic description of tamponade without circulatory compromise and hypoperfusion is not equivalent to generalized ischemia. When retrospectively applied to their own patient population (221 patients undergoing acute surgical repair in the 1993–2004 period), the in-hospital mortality was very distinctly predicted by the classification: 3.1 % in patients without any ischemia; 26 % with localized ischemia; 18 % with generalized ischemia and 40 % with localized and generalized ischemia. As pointed out by the authors in their descriptive report, the findings need to be validated, preferably in a prospective, multicenter setting to allow a broader application. Analyzing our recent 20-year experience with acute type A aortic dissection [18], we applied the Penn classification to 360 consecutively operated patients, and found (1) a very similar distribution between groups (60 % Penn Aa, 14 % Penn Ab, 18 % Penn Ac and 8 % Penn Abc), (2) a similar pattern of in-hospital mortality (14 % Penn Aa, 24 % Penn Ab, 24 % Penn Ac and 44 % Penn Abc), (3) statistically significant relationships between Penn class Ac and Abc and intraoperative death (Odds ratio 5.0 and 5.4, respectively), and between Penn class Abc and non-class Aa and in-hospital mortality (Odds ratio 3.4 and 2.3, respectively) in multivariable analysis. We concluded that the Penn classification is easy to adapt, report and interpret and, as suggested by the Penn group, in combination with the dissection extent according to the DeBakey-classification [26], adequately describes the important features of acute type A aortic dissection, rendering organ-specific break-down of malperfusion syndromes unnecessary and underscoring the role of tamponade and preoperative circulatory state as impacting on outcome only when resulting in clinical—generalized—ischemia.

The patient in the case presentation somewhat eludes the Penn classification, turning from an uncomplicated class Aa into a generally ischemic class Ac within a matter of minutes. Nevertheless, the classification is useful clinically as well as in reporting outcomes.

References

1. Song JK, Kang SJ, Song JM, Kang DH, Song H, Chung CH, Lee JW, Song MG. Factors associated with in-hospital mortality in patients with acute aortic syndrome involving the ascending aorta. Int J Cardiol. 2007;115(1):14–8.
2. Uchida K, Imoto K, Takahashi M, Suzuki S, Isoda S, Sugiyama M, Kondo J, Takanashi Y. Pathologic characteristics and surgical indications of superacute type A intramural hematoma. Ann Thorac Surg. 2005;79(5):1518–21.
3. Van Arsdell GS, David TE, Butany J. Autopsies in acute type A aortic dissection. Surgical implications. Circulation. 1998;98(19 suppl):II-299–302.
4. Ehrlich M, Fang C, Grabenwöger M, Cartes-Zumelu F, Wolner E, Havel M. Perioperative risk factors for mortality in patients with acute type A aortic dissection. Circulation. 1998;98(supp):II-294–8.
5. Bayegan K, Domanovits H, Schillinger M, Ehrlich M, Sodeck G, Laggner AN. Acute type A aortic dissection: the prognostic impact of perioperative cardiac tamponade. Eur J Cardiothorac Surg. 2001;20(6):1194–8.
6. Tan MESH, Kelder JC, Morshuis WJ, Schepens MAAM. Risk stratification in acute type A dissection: proposition for a new scoring system. Ann Thorac Surg. 2001;72(6):2065–9.
7. Mehta RH, Suzuki T, Hagan PG, Bossone E, Gilon D, Llovet A, Maroto LC, Cooper JV, Smith DE, Armstrong WF, Nienaber CA, Eagle KA, IRAD Investigators. Predicting death in patients with acute type A aortic dissection. Circulation. 2002;105(2):200–6.
8. Chiappini B, Schepens M, Tan E, Dell'Amore A, Morshuis W, Dossche K, Bergonzini M, Camurri N, Bacchi Reggiani L, Marinelli G, Di Bartolomeo R.

Early and late outcomes of acute type A aortic dissection. Analysis of risk factors in 487 consecutive patients. Eur Heart J. 2005;26(2):180–6.
9. Rampoldi V, Trimarchi S, Eagle KA, Nienaber CA, Oh JK, Bossone E, Myrmel T, Sangiorgi GM, De Vincentiis C, Cooper JV, Fang J, Smith D, Tsai T, Raghupaty A, Fattori R, Sechtem U, Deeb MG, Sundt III TM, Isselbacher EM, IRAD Investigators. Simple risk models to predict surgical mortality in acute type A aortic dissection: the international registry of acute aortic dissection score. Ann Thorac Surg. 2007;83(1):55–61.
10. Santini F, Montalbano G, Casali G, Messina A, Iafranscesco M, Battista Luciani G, Rossi A, Mazzucco A. Clinical presentation is the main predictor of in-hospital death for patients with acute type a aortic dissection admitted for surgical treatment: a 25 years experience. Int J Cardiol. 2007;115(3):305–11.
11. Gilon D, Mehta RH, Oh JK, Januzzi Jr JL, Bossone E, Cooper JV, Smith DE, Fang J, Nienaber CA, Eagle KA, Isselbacher EM, IRAD Investigators. Characteristics and in-hospital outcomes of patients with cardiac tamponade complicating type A acute aortic dissection. Am J Cardiol. 1029;103(7).
12. Girdauskas E, Kuntze T, Borger MA, Falk V, Mohr F-W. Surgical risk of preoperative malperfusion in acute type A aortic dissection. J Thorac Cardiovasc Surg. 2009;138(6):1363–9.
13. Goda M, Imoto K, Suzuki S, Uchida K, Yanagi H, Yasuda S, Masuda M. Risk analysis for hospital mortality in patients with acute type A aortic dissection. Ann Thorac Surg. 2010;90(4):1246–50.
14. Hansen MS, Nogareda GJ, Hutchison SJ. Frequency of and inappropriate treatment of misdiagnosis of acute aortic dissection. Am J Cardiol. 2007;99(6):852–6.
15. Blankenship JC, Almquist AK. Cardiovascular complications of thrombolytic therapy in patients with a mistaken diagnosis of acute myocardial infarction. J Am Coll Cardiol. 1989;14(6):1579–82.
16. Marian AJ, Harris SL, Pickett JD, Campbell E, Fromm RE. Inadvertent administration of rtPA to a patient with type 1 aortic dissection and subsequent cardiac tamponade. Am J Emerg Med. 1993;11(6):613–5.
17. Kamp TJ, Goldschmidt-Clermont PJ, Brinker JA, Resar JR. Myocardial infarction, aortic dissection, and thrombolytic therapy. Am Heart J. 1994;128(6 Pt 1):1234–7.
18. Olsson C, Hillebrant CG, Liska J, Lockowandt U, Eriksson P, Franco-Cereceda A. Mortality in acute type A aortic dissection: validation of the Penn classification. Ann Thorac Surg. 2011;92(4):1376–82.
19. Isselbacher EM, Cigarroa JE, Eagle KA. Cardiac tamponade complicating proximal aortic dissection. Is pericardiocentesis harmful? Circulation. 1994;90(5):2375–8.
20. Svensson LG. Editorial comment. Circulation. 1998;98(19 suppl):II-302–4.
21. Noera G, Lamarra M, Guarini S, Bertolini A. Survival rate after early treatment for acute type-A aortic dissection with ACTH-(1–24). Lancet. 2001;358(9280):469–70.
22. Khaladj N, Shresta M, Peterss S, Strueber M, Karck M, Pichlmaier M, Haverich A, Hagl C. Ascending aortic cannulation in acute aortic dissection type A: the Hannover experience. Eur J Cardiothorac Surg. 2008;34(4):792–6.
23. Kazui T, Washiyama N, Bashar AH, terada H, Suzuki T, Ohkura K, Yamashita K. Surgical outcome of acute type A aortic dissection: analysis of risk factors. Ann Thorac Surg. 2002;74(1):75–81.
24. Long SM, Tribble CG, Raymond DP, Fiser SM, Kaza AK, Kern JA, Kron IL. Preoperative shock determines outcome for acute type A aortic dissection. Ann Thorac Surg. 2003;75(2):520–4.
25. Augoustides JGT, Geirsson A, Szeto WY, Walsh EK, Cornelius B, Pochettino A, Bavaria JE. Observational study of mortality risk stratification by ischemic presentation in patients with acute type A aortic dissection: the Penn classification. Nat Clin Pract Cardiovasc Med. 2009;6(2):140–6.
26. Augoustides JGT, Szeto WY, Desai ND, Pochettino A, Cheung AT, Savino JS, Bavaria JE. Classification of acute type a dissection: focus on clinical presentation and extent. Eur J Cardiothorac Surg. 2011;39(4):519–22.