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STATE-OF-THE-ART PAPER

Evidence, Lack of Evidence, Controversy, and Debate in the Provision and Performance of the Surgery of Acute Type A Aortic Dissection

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Acute type A aortic dissection is a lethal condition requiring emergency surgery. It has diverse presentations, and the diagnosis can be missed or delayed. Once diagnosed, decisions with regard to initial management, transfer, appropriateness of surgery, timing of operation, and intervention for malperfusion complications are necessary. The goals of surgery are to save life by prevention of pericardial tamponade or intra-pericardial aortic rupture, to resect the primary entry tear, to correct or prevent any malperfusion and aortic valve regurgitation, and if possible to prevent late dissection-related complications in the proximal and downstream aorta. No randomized trials of treatment or techniques have ever been performed, and novel therapies—particularly with regard to extent of surgery—are being devised and implemented, but their role needs to be defined. Overall, except in highly specialized centers, surgical outcomes might be static, and there is abundant room for improvement. By highlighting difficulties and controversies in diagnosis, patient selection, and surgical therapy, our over-arching goal should be to enfranchise more patients for treatment and improve surgical outcomes. (J Am Coll Cardiol 2011;58:2455–74) © 2011 by the American College of Cardiology Foundation

Acute type A aortic dissection (ATAAD) is highly lethal and might be increasing in incidence (1). Surgery is believed to save and extend life, but despite apparent advances, diagnosis is often delayed, evidence for improving outcomes is modest (2), and optimal surgical management remains unclear (3–8). Recent reviews (6,7,9–12) have directed limited attention to the provision and performance of surgery. This review specifically examines areas of uncertainty and controversy in diagnosis, provision of care, and surgical management of ATAAD and has the objectives of promoting investigation to detect and treat the condition earlier and to improve treatment outcomes.

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Anatomy and Incidence

By definition, ATAAD involves the ascending aorta (AscAo) and includes types I and II of the DeBakey classification. The DeBakey classification categorizes dissections as type I (involving the AscAo with distal extension), type II affecting the AscAo only, and type III delineating disease of the descending aorta (DescAo) and beyond. Modifications of these classifications—which describe patho-anatomical variants—exist, but for practical purposes, the original classifications by specifying AscAo involvement and hence risk of intra-pericardial rupture define those cases requiring surgery (Fig. 1).

Neither classification dictates the site of the originating entry tear. In ATAAD a primary intimal tear is usually present within the AscAo, sometimes accompanied by secondary, more distal tears (13). In others, involvement of the AscAo is due to retrograde propaga-

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Abbreviations and Acronyms

ARR = aortic root replacement

AscAo = ascending aorta (or aortic)

AscAoR = ascending aorta (or aortic) repair

ATAAD = acute type A aortic dissection

CAD = coronary artery disease

CT = computed tomography

CTn = cardiac troponin

DescAo = descending aorta

ECG = electrocardiogram

FemA = femoral artery (or arterial)

FET = frozen elephant trunk

FL = false lumen (or luminal)

HCA = hypothermic circulatory arrest

IRAD = International Registry of Aortic Dissection

ODA = open distal anastomosis

PFL = patent or persistent false lumen

RAxA/ScA = right axillary artery (arterial)/subclavian artery (arterial)

TAR = total aortic arch replacement

TEE = transesophageal echocardiography

TL = true lumen (or luminal)

VSRR = valve-sparing root replacement

tion of a dissection with a primary tear originating within the arch or DescAo (14). On occasion, no intimal tear is identifiable. Other acute aortic syndromes, intramural hematoma, and deep penetrating ulcer might have similar presentations (15). The estimated total incidence of acute (type A and B) dissection is 30 to 43/1 million of population (pmp)/year (16,17). The ATAAD constitutes >50% of all cases (6), and DeBakey type I lesions predominate. In the absence of predisposing conditions, ATAAD is more common with increasing age. It is not known whether the apparent increase in incidence represents improved rates of diagnosis or a real effect due to changing population age and risk profiles (Fig. 2) (1,18).

Are We Diagnosing and Treating All Patients?

Acute type A aortic dissection has a plethora of presentations that generate diagnostic uncertainty. Although chest pain is the most commonly reported symptom, it might be absent in 15% to 20% of cases (19). Clinical diagnostic suspicion is mandatory (12,20), because individual signs, symptoms, electrocardiogram (ECG), and chest radiograph changes lack both sensitivity and specificity (5). Without clinical suspicion, patients are not immediately channeled into an appropriate imaging pathway. Most diagnostic chest pain algorithms, primarily designed to detect acute coronary syndrome (ACS), do not prompt imaging unless ATAAD is suspected and the application of triple rule-out coronary angiographic computed tomography (CT) is likely to be limited and again based on clinical suspicion (21–23). Therefore, ATAAD diagnosis is rarely immediate and might be missed, misdiagnosed, or substantially delayed in up to 40% of cases (24–26), sometimes being only established at autopsy (13,15,25).

The ECGs and cardiac troponin (CTn) estimations are primarily used to facilitate ACS diagnosis and do not discriminate between ACS and dissection. The ACS-type ECG findings occur in 26% of patients with ATAAD (Online Table 1) (27) and, when present with CTn elevation, might lead to misdiagnosis and inappropriate therapy, including antiplatelet agents, heparin, and thrombolysis. Acute aortic syndromes have no reliable point-of-care biomarkers. Several have been investigated (28,29), but none are sufficiently discriminant for general use. At present, D-dimer measurement might be the most useful (30–35). A negative D-dimer assay is highly predictive that a patient does not have dissection, whereas a high level makes the differential diagnosis of ATAAD or pulmonary embolism far more likely (Fig. 3) (35). Both diagnoses might require confirmatory cross-sectional imaging. If the number of patients with ATAAD that can be successfully identified and treated is to be increased, much work is required to discover and validate existing and novel biomarkers.

Once suspected, definitive imaging comprising CT, transthoracic echocardiography and transesophageal echocardiography (TEE) and magnetic resonance imaging are required for confirmation (6,7,36). The diagnostic accuracy of these investigations has been reviewed elsewhere (5). Multislice ECG-gated CT angiography is likely to improve diagnostic precision (37).

ATAAD Outcomes With and Without Surgery

Many ATAAD patients die, either before antemortem diagnosis or treatment, due to aortic rupture, tamponade, malperfusion phenomena, and heart failure secondary to aortic regurgitation or coronary malperfusion.

The untreated mortality of type A dissection has been reported to be approximately 1% to 2%/h after symptom onset, with up to 90% of patients succumbing within 30 days (38–40). In some of the earliest reports, 24-h mortality was >35%, and over one-half the patients had died within 48 h (41–44).

If diagnosed, surgical repair is possible (26) but remains high-risk, with both mortality and neurological complication rates of 15% to 30%, despite increased understanding of the pathophysiology, improved anesthetic, myocardial and cerebral protection techniques, and advances in postoperative care (39,45–48). No randomized studies of medical versus surgical management have ever been performed, but on the available evidence, surgery converts a 90% mortality risk to at least a 70% survival chance. This magnitude of effect means that ATAAD is an accepted indication for surgery (4–7,39,46,49), and no more than 2 patients need to be treated to gain survival benefit. The survival advantage of surgery versus medical management continues in the longer term (50,51) as survival curves continue to diverge.

Despite a number of laudable individual center series, the evidence that overall surgical outcomes are improving is modest, and the mortality rates of 15% to 30% reported in the last 2 decades of the 20th century might not be improving in the new millennium (2,46–48,52).

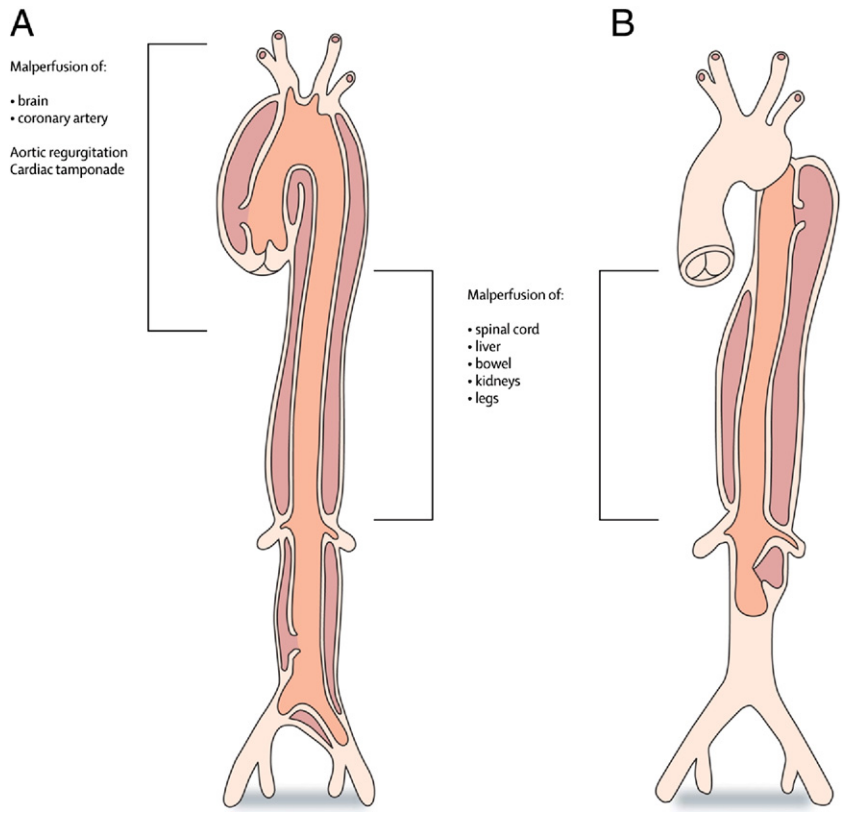

Figure 1 **Subtypes and Complications of Aortic Dissection**

(A) Type A and **(B)** Type B aortic dissections. Type A encompass DeBakey I (ascending aorta only and, therefore, less potential for malperfusion phenomena) and II and Type B DeBakey III classifications. Reproduced, with permission, from Golledge and Eagle (12).

Although the Stanford classification conveniently compartmentalizes AscAo involvement and treatment direction, the DeBakey classification provides more prognostic information. Perioperative, long-term, and aneurysm-free patient survival is greater in Type II than Type I dissection. This relates to the propensity for distal malperfusion phenomena and persistence of a distal false lumen (FL).

Patient selection for surgery: should all ATAAD patients be operative candidates? Some ATAAD patients have such advanced presentations, particularly due to malperfusion phenomena, that surgical repair seems futile. The pre-operative presentation of coma or in a collapsed state with shock secondary to either pericardial tamponade or coronary dissection and ischemia are consistent predictive factors for postoperative mortality (39,53–61). These and other factors have been incorporated into predictive risk models (56,57) on the basis of International Registry of Aortic Dissection (IRAD) and individual center data (Tables 1, 2, and 3) (46,62), which might aid the decision-making of the attending surgeon (46). However, although each complication might engender additional risk, this does not preclude a superior outcome with surgery and rigid treatment exclusion criteria are inappropriate. Nevertheless, certain patients, moribund or comatose due to the lethal consequences of established malperfusion, might be excessively high-risk operative candidates, and selected medical management, dictated by local center outcomes for such cases, is justifiable.

ATAAD repair in octogenarians. In Western societies, nearly one-fifth of the population are >65 years of age, and this fraction is increasing. The ATAAD incidence increases with age, and as with all emergency cardiovascular surgery, age is an independent predictor of worse operative mortality and morbidity and reduced longer-term survival (38,39,47,55–59,61,63–68). Data from the IRAD registry suggest that one-third of patients presenting with ATAAD are over 70 years of age, with only 47.6% of patients older than 80 years of age undergoing surgery. Surgical mortality for this group approximates 40%, compared with 58% for medically managed patients (69). In some series, surgery on octogenarian ATAAD patients is associated with a 40% to 80% perioperative mortality (70,71). In contrast, survival and hospital discharge rates of 41% have been reported in some medically treated patients (39). Such reports and the significant mortality and neurological event risk evident even within younger patients has prompted a debate with

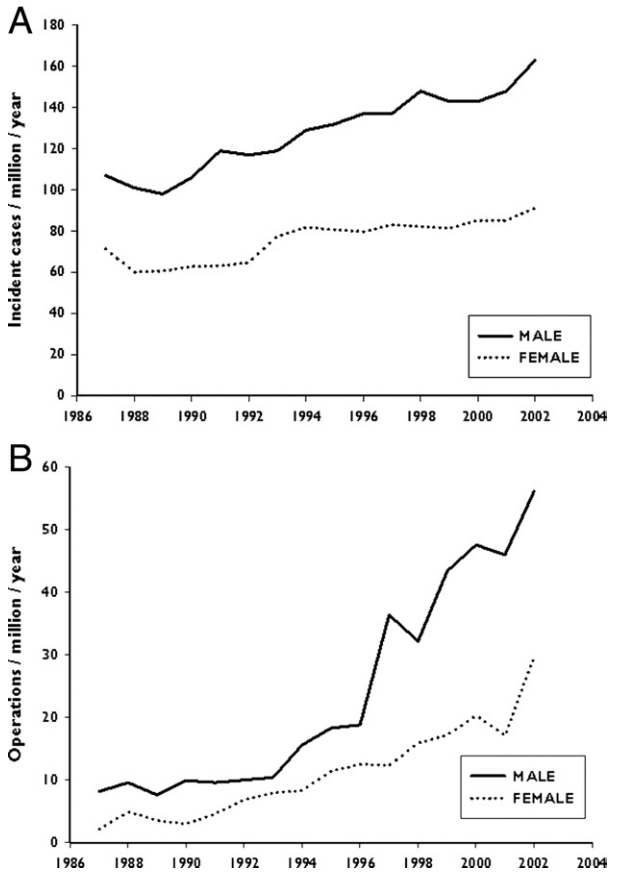

Figure 2 **Increasing Incidence of Aortic Aneurysm and Dissection Over a 15-Year Period**

Reproduced, with permission, from Olsson et al. (1).

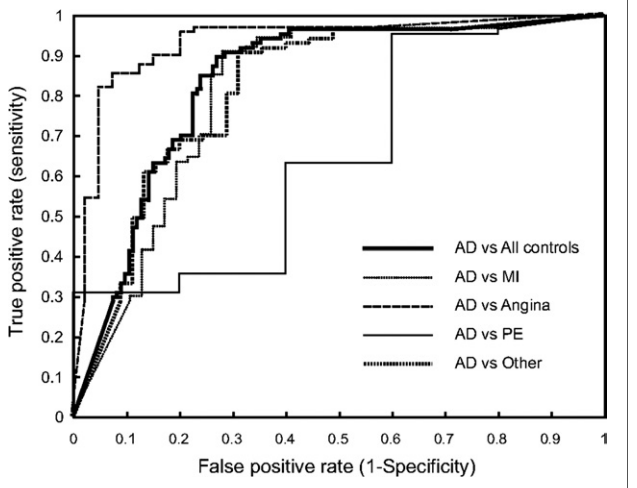

Figure 3 **ROC to Demonstrate the Performance of D-Dimer Analysis in the Diagnosis of Patients With Acute AD**

The area under the curve on receiver-operator characteristics (ROC) curve analysis for patients with aortic dissection (AD) within 24 h of symptom onset versus all control subjects was 0.84 (95% confidence interval [CI]: 0.78 to 0.89) and 0.81 (95% CI: 0.72 to 0. 90), 0.93 (95% CI: 0.87 to 0.98), 0.65 (95% CI: 0.36 to 0.93), and 0.82 (95% CI: 0.73 to 0.90) versus myocardial infarction (MI), angina, pulmonary embolism (PE), and other uncertain diagnoses, respectively. The greatest area under the ROC curve is for AD versus angina; lower values are found for MI, and D-Dimer is unable to differentiate AD from PE but could direct patients into an appropriate imaging algorithm. Reproduced, with permission, from Suzuki et al. (35).

regard to the appropriateness of surgery in octogenarians (70,72–79). The data are conflicting. Some expert groups have demonstrated excellent survival with few complications (72,77,80–82) and maintained quality of life (83,84), outcomes highly superior to those anticipated with medical management (85,86). However, a proportion of surviving patients might be significantly compromised and unable to live independently (74). Data from the IRAD registry at present show ATAAD repair in octogenarians remains reasonable, provided the additive risk assessment of malperfusion complications and pre-existent comorbidities are considered and clinical judgment in light of local surgical outcomes is applied.

Where should surgery be performed? The ATAAD surgery, although conceptually simple, is actually practically demanding. Difficulties arise in the handling of tissues profoundly weakened by dissection; the judgments; and the need to anticipate, identify, and react to any intra-operative malperfusion. Most (approximately 75%) ATAAD patients are diagnosed in peripheral hospitals (87) and transferred to the nearest surgical center for treatment. Few such centers have established programs of aortic surgery, and fewer still will have comprehensive on-call arrangements for multidisciplinary aortic teams, including surgeons, anesthesiologists, and endovascular specialists. Thus, ATAAD patients might undergo surgery by a team unfamiliar both with the condition and the adjunctive techniques that are believed to affect outcome (e.g., varied arterial cannulation techniques, techniques of aortic valve reconstruction or aortic root replacement [ARR], adjunctive cerebral protection techniques, extended procedures and interventions for malperfusion).

This unfamiliarity provides a cogent argument for centralizing dissection and thoracic aortic surgery management. This would concentrate personnel, expertise, and systems, allowing patients to benefit via the volume-outcome rela-

Table 1 **Preoperative Predictors of Mortality Associated With Type A Dissection**

Variable	Death Odds Ratio (95% CI)
Age ≥70 yrs	1.98 (1.19–3.29)
History of aortic valve replacement	4.21 (not available)
Presentation with hypotension, shock or tamponade	3.23 (1.95–5.37)
Migrating chest pain	2.42 (1.32–4.45)
Pre-operative tamponade	2.65 (1.48–4.75)
Any pulse deficit	1.75 (1.06–2.88)
ECG infarction or new ischemia	1.76 (1.02–3.03)

Adapted and reproduced, with permission, from Rampoldi et al. (57).
CI = confidence interval; ECG = electrocardiogram.

Table 2 Intra-Operative Predictors of Mortality Associated With Type A Dissection

Variable	Death Odds Ratio (95% CI)
Age ≥70 yrs	1.79 (1.02–3.15)
History of aortic valve replacement	5.93 (2.07–16.97)
Presentation with hypotension, shock, or tamponade	2.52 (1.40–4.54)
Migrating chest pain	2.02 (1.02–4.02)
Any pulse deficit	1.90 (1.10–3.29)
In operation	
Hypotension or shock	3.81 (2.16–6.71)
RV dysfunction	4.90 (2.00–12.00)
Partial arch	0.52 (0.28–0.98)
CABG	2.54 (1.23–5.24)

Adapted and reproduced with permission from Tan et al. (62).
CABG = coronary artery bypass grafting; CI = confidence interval; RV = right ventricular.

tionship consistently demonstrated in cardiovascular surgery (88–90). However, although performance and quality metrics are in widespread use in other areas of cardiovascular disease, none have as yet been established for thoracic aortic surgery or ATAAD management (23). Where geography and transport times allow, peripheral hospital presentation and diagnosis provides an opportunity to have directed transfer to specialist-designated centers (91,92). Such directed transfer could improve outcomes significantly and would be enhanced if biomarkers were identified that allowed detection of patients with a high risk of dissection at pre-hospital presentation.

When should surgery be performed? Because the untreated natural history is so lethal, the possibility of directed transfer has to be reconciled against time to treatment. In 1934, Shennan (93) reported that 40% of ATAAD patients die immediately, 70% within 24 h, 94% within 1 week, and 100% within 5 weeks. Similar attrition rates have been reported by others (41,43,94–97) and are the justification for emergent transfer policies to the nearest available center (7,9,12). Although the need for emergency surgery is apparent, it should be noted that the reported high mortality rates in early series included patients who died abruptly at symptom onset (41) in whom diagnosis was only made at postmortem. More recent data suggest that the 30-day attrition rate for patients admitted and diagnosed in life but not undergoing surgery is actually lower and slower than first thought (39,57,98–100). The risk of death seems to exponentially reduce as the first hours after symptom onset pass. In diagnosed cases, time from initial symptoms to hospital presentation approximates 1 to 2 h (87). Delayed admission adversely affects outcome (101). Once admitted, the time to diagnosis varies greatly (102); 50% of patients have a time to diagnosis >6 h in Europe and >15 h in the United States, with 75% of patients having diagnostic times >3 to 4 h (87). A time-dependent post-admission mortality risk can be inferred from IRAD and other published data. A medically managed ATAAD patient has a 15% to 30% mortality risk in the first 24 h of admission, a 10% to 20% risk between 24 and 48 h, and a similar risk between days 2 and 5 (39,43,44,51). Between days 5 and 30, the risk of death reduces to approximately 1%/day (Online Fig. 1) (51).

Diagnostic delay is increased by presentations that do not evoke clinical suspicion, including painless onset, dyspnea secondary to heart failure or pleural effusion, CTn-positivity, ACS-type ECG, neurological presentations, limb ischemia, or abdominal pain (12,103,104). The time from symptom onset to commencement to surgery is even longer, and contrary to expectations, 20% to 50% of patients do not undergo surgery until 24 h or more after symptom onset (51,104,105). In some series, nearly one-half of all patients have a delay of more than 48 h between symptoms and presentation to a tertiary center (104).

Because the risk of death between symptom onset and 48 h is appreciable, emergency operation remains indicated in patients presenting during this period. Early presenting, apparently stable ATAAD patients have an ongoing mortality risk in the interval between center admission and operation, and this might constitute 5% of admitted cases (104). No report suggests delay in the first 24 h after symptom onset is safe practice, and immediate emergency status should be accorded to those rapidly diagnosed patients presenting early after symptom onset with hemodynamic compromise, shock, tamponade, myocardial ischemia, or heart failure secondary to acute aortic valve regurgitation or neurological disturbance. Pre-operative tamponade is an important risk factor for mortality, and because presentation for the majority of cases occurs outside specialist centers, a decision with regard to pericardiocentesis for stabilization is necessary, but the appropriateness of this is debated (106).

The optimal management of ATAAD patients arriving at a center beyond 48 h of symptom onset who are pain-free and hemodynamically stable without evidence of malperfu-

Table 3 Independent Pre-Operative Risk Factors for Mortality in Patients With Acute Type A Aortic Dissection

Variable at Presentation	% Incidence in Type A	% Among Survivors	% Among Nonsurvivors	Mortality Odds Ratio (95% CI)
History of aortic valve replacement	4.4	3.5	7.4	3.12 (1.16–8.40)
Migrating chest pain	14.2	12.1	20.5	2.77 (1.49–5.15)
Presenting hypotension	17.6	13.3	30.4	1.95 (1.08–3.52)
Presenting shock/tamponade	24.7	19.5	40.7	2.69 (1.41–5.11)
Pre-operative cardiac tamponade	15.7	11.8	27.6	2.22 (1.17–4.22)
Pre-operative limb ischemia	9.7	7.8	15.8	2.10 (1.00–4.38)

Modified and reproduced, with permission, from Trimarchi et al. (46).
CI = confidence interval.

sion or pericardial effusion is unclear. Such patients are likely to be older with more comorbid disease and are more likely to present with evidence of heart failure and abrupt chest pain and have arch involvement (104). Although a continued attrition rate is recognized, there is perhaps sufficient time to undertake additional investigations and schedule surgery urgently but not emergently. This approach should be aborted by any evidence of recurrent pain or instability. In a nonrandomized study, equivalent outcomes were obtained when such patients were more fully investigated before planned daytime urgent surgery, but the impact of additional investigations on management is unclear, and selection bias is possible (104). Thus, at present, ATAAD within 48 h of symptom onset is a surgical emergency. Stable patients presenting to surgical centers beyond this time might potentially be scheduled in a more planned fashion.

Malperfusion

Malperfusion phenomena have an incidence of 16% to 33% (100,107–110) and might result in myocardial, cerebral, spinal, extremity, renal, and visceral ischemia. They are most common in extensive DeBakey type I dissection (12,111). Clinically apparent malperfusion of any type increases mortality risk (107,109,111,112). Although demonstration of radiological malperfusion might also be important, it is the ischemic consequences of malperfusion and end-organ dysfunction that compromise survival (Fig. 4) (100,111,113,114). Thus, although a patient might have CT evidence of reduced true luminal (TL) innominate artery flow, the prognostic relevance of this is primarily determined (Online Table 2) by the presence of clinical neurological ischemia.

The mechanism is variable; malperfusion might occur due to fixed or dynamic flap occlusion of the aorta or branch artery or secondary to compromised flow in the supplying FL or TL due to thrombosis of the former and compression of the latter (112). The TL can also be significantly compressed by a higher pressure FL, due to large entry tear and an absent or small exit tear. Intimal shearing with flap occlusion or intussusception within the aorta or its branches might also occur.

Coronary malperfusion. Approximately 10% to 15% of cases display coronary malperfusion (115,116). Over one-half of these are initially misdiagnosed as ACS and might inappropriately receive antiplatelet or thrombolytic therapy. An ACS-type ECG is associated with more complicated dissections and independently predicts early mortality (27). Right coronary artery involvement is more frequent, and any coronary malperfusion adversely affects outcome (56,117–119). The sinus of Valsalva pathology and dissection involvement associated with coronary malperfusion increases the likelihood that ARR will be necessary (45,116,120).

The incidence of coronary artery disease (CAD) in ATAAD is generally believed to be low, although 1 study suggested that 1 in 3 patients will have evidence of CAD (121). Notwithstanding this, there is a consensus that coronary angiography should not be performed in the emergency setting (122–124), because yield is low, emergency operative intervention might be delayed, and catheterization could lead to aortic rupture or malperfusion exacerbation. Currently, coronary ischemia in the emergent patient, without known CAD, should be assumed to rep-

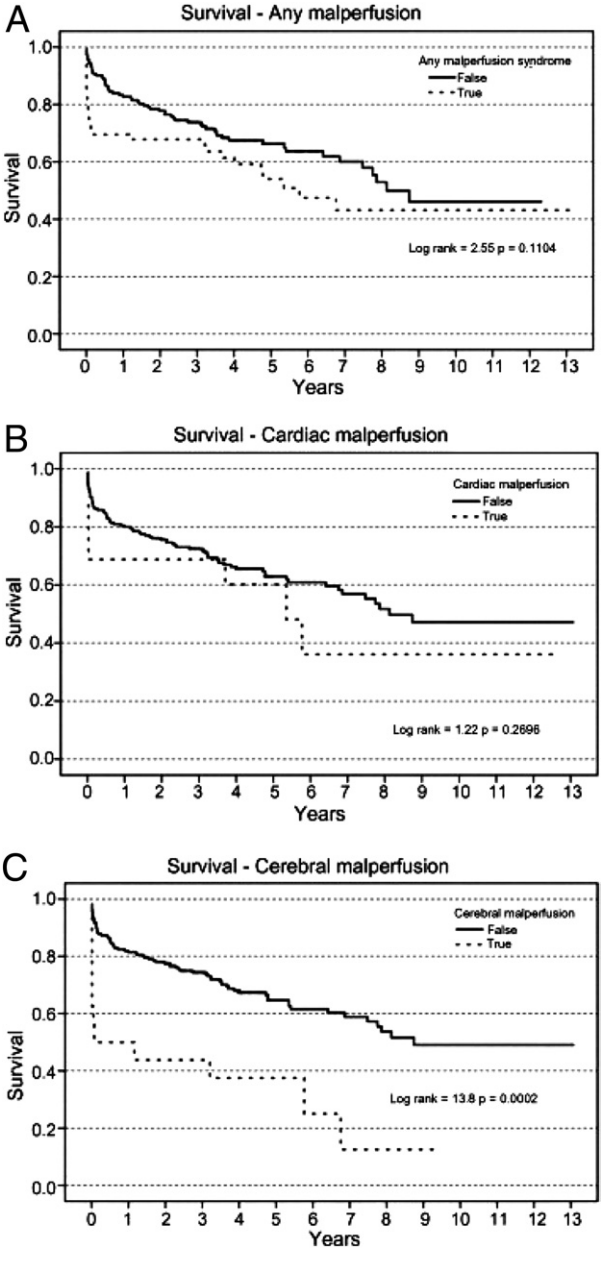

Figure 4 Kaplan-Meier Survival Curves of Actuarial Survival

Kaplan-Meier survival curves of actuarial survival of patients with **(A)** any malperfusion syndrome, **(B)** coronary malperfusion syndrome, and **(C)** central nervous system malperfusion syndrome. Reproduced, with permission, from Geirsson et al. (120).

resent a malperfusion phenomenon. Coronary angiography is reserved for stable patients with a clinical history of CAD or intervention and perhaps those presenting >48 h after symptom onset (104). Intra-operative angiography has been suggested but is not yet in common use (125,126). An alternative strategy would be the use of ECG-gated CT angiography to assess for the presence of CAD.

The surgical technique required to deal with coronary malperfusion depends upon its mechanism, which might be embarrassment of the coronary ostium by a dynamic intimal flap, dissection of the ostium with intimal shearing and even intimal intussusception (117,120,127–129). Compromised flow due to TL compression with an intact intima can be dealt with by re-affixation of the dissected layers, even if there is periostial dissection. Alternatively, coronary ostial button re-implantation might be performed if root replacement has been required. The classical Bentall procedure—in which ostial re-implantation is performed without button creation—is ill-advised, because the risk of anastomotic disruption and pseudoaneurysm is increased. For more extensive vessel dissection or intimal disruption, coronary artery bypass grafting—performed in approximately 15% of cases—is necessary, usually with autologous saphenous vein. The threshold for coronary artery bypass grafting should be low.

In the presence of coronary malperfusion, in-hospital mortality doubles and some patients will die intra-operatively from low cardiac output. Longer-term survival is also reduced with an overall 5-year survival of 22% (116). However, if hospital survival is achieved, conditional survival thereafter is nearly equivalent to those without coronary malperfusion.

The management of brain malperfusion and paraplegia. Brain malperfusion is usually due to TL flow embarrassment of a supra-aortic artery by FL pressurization. Its management remains a surgical dilemma. Patients with dense deficits or coma have a poor post-operative prognosis, and intervention in such cases might be considered futile (46,113,116,130). However, recovery has been reported in isolated cases in which rapid reperfusion is achieved (131–135). In contrast, conscious patients with a transient or even persistent neurological deficit pre-operatively have a similar operative and longer-term survival as nondeficit patients, and over 50% will fully regain neurological function (116,120). The timing of intervention is crucial; a shorter time between symptom onset and surgical reperfusion improves outcome, and if reperfusion is achieved in <5 h, this has a reasonable prospect of limiting stroke progression (136,137). A precise cutoff time above which recovery is unlikely is undefined. A 10-h threshold below which neurological conservation and recovery might be higher has been suggested (116,138), but reperfusion is no guarantee that stroke will be prevented or that full independent rehabilitation will occur (111,116,139). Because speed of reperfusion is so important, early presenting ATAAD with brain malperfusion should have direct transfer to the operating room. Cannulation and reperfusion strategies might be critical in improving neurological outcome, but it should be noted that high de novo neurological complication rates of ATAAD repair remain a significant risk (140).

Paraplegia due to malperfusion phenomenon might also occur, but its etiologic mechanism is less clear, due to the multi-source origin of cord perfusion. Shearing of multiple intercostal arteries might abruptly interrupt anterior spinal artery collaterals, and this might be exacerbated by any embarrassment of vertebral artery flow, dynamic alterations in aortic TL flow, or compromise of internal iliac perfusion. Not all of these might be correctable by restoration of TL flow, and in consequence, the prospects for recovery are less; there are only a limited number of successful paraplegia reversals (116,120). However, in the hope rather than anticipation of neurological recovery, early presenting paraplegia represents an indication for emergency intervention.

Extremity malperfusion. Limb malperfusion (most commonly lower limb) occurs in approximately 12% of ATAAD patients. As the primary presentation, it is a cause of diagnostic delay, particularly when unaccompanied by torso pain. Although often accompanied by malperfusion phenomena elsewhere, in isolation, limb malperfusion does not affect survival outcome. A higher incidence of acute renal failure post-operatively, presumably related to reperfusion nephrotoxin release and contrast investigations, has been noted (116). Standard surgical therapy with restitution of orthograde TL flow successfully resolves limb malperfusion in most cases, but some will require additional femoro-femoral bypass or endovascular procedures, including intimal flap fenestration or stent-graft placement if malperfusion persists after the primary repair (141).

Visceral malperfusion. Of all such phenomena, visceral malperfusion is the most lethal, heralding an extremely poor prognosis, unless the malperfusion is corrected and nonviable gut is excised (110). Advanced visceral malperfusion, managed by initial ascending aortic surgical repair and post-repair laparotomy, has a high mortality (100,142). This mortality might reduce if malperfusion is first treated by endovascular fenestration with or without branch vessel stenting (100,113,143). However, delay in ascending aorta repair (AscAoR) leads to a significant attrition rate, due to aortic rupture (113,144). Patient survival is thus dependent upon both the duration, extent, severity, and reversibility of visceral ischemia and the ability to repair the AscAo. Therefore, management is primarily an issue of diagnostic timing. Surgical opinion is divided, with some advocating initial percutaneous or extra-anatomic reperfusion (145) of either clinically significant or radiologically identified malperfusion followed by delayed AscAo repair and others recommending immediate AscAo repair rapidly followed by investigation and treatment of residual malperfusion. Another option, which requires hybrid operating theaters, comprises near-contemporaneous percutaneous interventions or antegrade stent deployment at the same sitting as the proximal procedure (146–151), but whether angiographic pursuit of subclinical malperfusion and endovascular

reperfusion should precede or follow the proximal surgery is unclear. Initial interventional angiography might subject the patient to a risk of rupture and renal injury that might also adversely affect survival (46,144). Very early presentation might yet be best treated by early restitution of aortic TL flow by primary AscAoR.

The prevailing view is that, in the absence of ongoing severe visceral ischemia with gangrenous bowel and acidosis, proximal repair should occur first. If there is concern with regard to visceral perfusion on initial imaging, this can be verified via a laparotomy extension of sternotomy. However, when visceral ischemia is clinically apparent and advanced, peripheral reperfusion and resection of ischemic intestine as a preliminary course of action seems more likely to achieve patient survival and prevent futile aortic reconstruction in nonviable candidates (116). At present, there is insufficient evidence to declare which strategy is optimal in which patients, but again, the complexities of ATAAD management is a prompt toward transfer to designated multidisciplinary units equipped with a full array of interventional, hybrid, and surgical techniques (152).

Renal malperfusion responds well to primary aortic surgery, but some cases will require additional interventional procedures (116,120).

The Aims and Objectives of ATAAD Surgery

The primary aim is prevention of intra-pericardial rupture. This is accomplished by AscAoR accompanied, where possible, by excision of the proximal entry tear. Other objectives include prevention or treatment of coronary ostial dissection, correction of any aortic valve insufficiency, restoration of dominant TL flow in the distal aorta, correction of distal malperfusion, and if possible, permanent obliteration of the FL in the distal aorta (12). Techniques used to achieve these aims have not been subject to randomized studies (4).

Historically, various methods of AscAoR have been described, including inlay grafts, direct entry excision, and end-to-end aortic anastomosis as well as sutureless, ringed intra-luminal grafts (153–155). Most current approaches replace the AscAo with a prosthetic graft (usually protein-impregnated to prevent blood extrusion) and use sutured, often buttress-reinforced anastomoses (156).

Technical aspects remain issues of debate (157–159)—even in how anastomoses should be constructed (160,161). Anastomoses must be hemostatically secure, a great challenge in ATAAD surgery. Various surgeon-specific techniques—including no reinforcement, external buttressing, bi-layer buttressing, or fabric insertion in the dissection plane—have been advocated, but there are few comparative data to guide practice (157,162).

The site of arterial cannulation for bypass. Acute type A aortic dissection repair requires cardiopulmonary bypass necessitating arterial cannulation (Online Table 3). The femoral artery (FemA) has been used for decades, due to ease of access, size, and ability to achieve adequate flow (163,164). However, FemA perfusion carries the potential risk of differentially pressurizing the FL, generating malperfusion (165–167). In autopsy series, the theoretical potential for brain malperfusion with FemA cannulation was 42%, whereas perfusion via the axillary artery limited this to only 16% (13). However, the clinical incidence of malperfusion with FemA cannulation is low (168), provided intra-operative monitoring enables rigorous real-time evaluation of brain perfusion, allowing intra-operative adjustment of cannulation site (169). To monitor intra-operative brain malperfusion, arterial pressure monitoring both proximal and distal to the aortic arch is advised (129,170), supplemented by forehead regional near infra-red spectroscopic monitoring of cortical regional mixed oxygen saturation (171). No currently available device can survey perfusion adequacy of the whole brain. Intra-operative time-points at which brain malperfusion is a particular hazard include the initiation of bypass, placement of an AscAo clamp, and reinstitution of bypass after aortic repair. To ensure TL perfusion after distal reconstruction, to minimize retrograde delivery of embolic material, and to minimize FL pressurization, there is an overwhelming consensus that orthograde corporeal perfusion via the aortic graft should be instituted if FemA cannulation has previously been used. Femoral cannulation is ill-advised in the presence of distal aortic atherosclerotic disease, because it risks proximal atheroembolization (172,173). In the context of ATAAD, initial FemA cannulation remains reasonable, provided malperfusion monitoring is applied and aortic imaging confirms an absence of atheroma. In cases when adequate bypass flow is not achievable with FemA return, a secondary cannulation technique becomes mandatory. A FemA cannulation is contraindicated in the presence of known or suspected coarctation (e.g., ATAAD in Turner's syndrome).

The use of the right axillary or subclavian artery (RAxA/ScA) has been advocated as an alternative (174–178). Surgical preparation is more time-consuming, but several reports (179–183) suggest improved outcomes with RAxA/ScA cannulation in ATAAD and arch surgery, compared with historical controls (184). An RAxA/ScA cannulation, however, is not risk-free, and complications include brachial plexus injury and de novo or propagation of dissection (149,185–191), and the need for intra-operative brain monitoring remains important. Although RAxA/ScA cannulation reportedly might reduce stroke rate and mortality in ATAAD (180,192), published series have often compared outcomes with a sicker, more emergent FemA patient group. If stroke protection is truly reduced, this might relate to earlier perfusion of a compromised innominate artery, maintenance of antegrade brain blood flow during any arrest period, or the avoidance of retrograde flow in an atheromatous aorta (193). In the absence of randomized or carefully matched cohort studies, a uniform recommendation for the primary use of RAxA/ScA cannulation is not currently justified. In small series, combined RAxA/ScA and FemA

cannulation have been used to prevent malperfusion with encouraging outcomes (194). Carotid and innominate artery cannulation to avoid malperfusion have also been used by some authors (195–197,198), and occasionally unique perfusion solutions have been generated to combat intra-operative malperfusion (199).

The importance of ensuring TL perfusion has led to the usage of direct cannulation approaches, including passage of a cannula into the AscAo via the left ventricular apex (200). Advantages include speed, simplicity, adequacy of flow, and apparent reliability of TL perfusion. However, trans-apical cannulation carries the risk of ventricular injury, cannula malposition via an intimal tear into the FL, and intra-operative aortic regurgitation. Although a primary technique for some surgeons, it is an important secondary cannulation site for others, if malperfusion is detected during perfusion via another peripheral route.

Direct cannulation of the TL within the AscAo (194,201–207) is a further alternative. It can be achieved in 2 ways. Firstly, after venous drainage exsanguination, the AscAo can be partially transected, the TL can be identified, and a cannula can be sited under direct vision (206). A second method is to directly cannulate with ultrasound imaging or CT inspection to identify the true lumen (208). This method achieves outcomes equivalent to FemA cannulation, but debate over safety and efficacy continues (192,209). Because the superiority of specific cannulation techniques has not been established (164,192,193,210), currently the cannulation site should be selected on the basis of pathology, patient status, the presence of atheromatous disease or malperfusion, and surgical familiarity with the options available. For each case, the surgeon should be familiar with several alternatives.

Aortic clamping during the conduct of proximal repair. It is possible to clamp the distal AscAo while on bypass and undertake the proximal part of the repair (116,208). Although clamping could allow FL pressurization and malperfusion, retrospective series have not identified this as a significant risk (211–213), except possibly for neurological morbidity (214,215). However, many experienced aortic surgeons counsel against clamping, proceeding initially to the open distal reconstruction followed by the proximal surgery during the re-warming phase. Clamping during the cooling phase might still be necessary in the presence of ascending aortic rupture or to expedite treatment of coronary malperfusion or aortic regurgitation with left ventricular distension.

What procedure for the aortic valve and root? The intra-operative management of the ATAAD aortic root involves assessment of aortic valve competence, annular diameter, morphology, dimension of the sinuses of Valsalva and the sino-tubular junction, the coronary ostial position, and the extent of proximal dissection propagation (155). There is consensus that direct inspection should be supported by intra-operative TEE. New aortic regurgitation in ATAAD usually occurs when the dissection involves the aortic root detaching the commissures from the aortic wall, permitting cusp prolapse (216). Approximately 30% of patients have an aortic diastolic murmur, and approximately one-half have aortic regurgitation on investigation (216). The surgical management of this is controversial (217,218). Preservation of the native aortic valve has obvious advantages. Because valve leaflet morphology is often normal, valve competence can usually be restored by re-affixing the commissures to the aortic wall (219,220) (Online Fig. 2) with a nondissected commissure height as a reference. The technique uses full-thickness suture fixation, but this might be supplemented by glue fixation of apposed dissected layers (49,221–223) or insertion of custom-sized and -shaped fabric neomedia inserts (157,158,224). The method used is surgeon-specific and comparative data are not available. Glue fixation alone might not be secure, increasing the risk of re-operation (223,225,226). Flap interference with valve closure or central regurgitation due to prior aortic dilation at the sino-tubular junction are additional causes of regurgitation that might be amenable to valve-conserving repair.

Despite satisfactory early outcomes of conservative valve management (49,215,225,227,228), 20% to 25% of patients might develop late root enlargement or progression of aortic regurgitation, necessitating aortic valve replacement or ARR (105,229,230). Risk factors include an aortic annulus $\geq$27 mm at initial surgery and above-moderate valve regurgitation (225,230). Therefore, some centers use a more aggressive strategy with a low threshold for prosthetic ARR (Online Fig. 3) (231,232). This strategy reduces the risk of native valve failure but adds the risk of prosthetic valves. An ARR could also increase risk in inexpert hands. Thus, the role of aggressive ARR management of the aortic valve versus conservative valve re-suspension is incompletely defined.

Sinus-retaining, valve-conserving techniques are inappropriate in patients with pre-existing root pathology as in Marfan's syndrome or annulo-aortic ectasia (233). In such cases, ARR with a composite prosthesis is recommended. An alternative is a valve-sparing root replacement (VSRR) procedure (227,234). Of the 2 types of VSRR, the re-implantation technique might be superior in ATAAD (23,227,234,235). However, VSRR requires longer procedure durations and more surgical judgment and might be the right operation at the wrong time.

When a competent noncalcified bicuspid aortic valve is detected, the decision to conserve will depend upon patient age, presence of annulo-aortic ectasia, and degree of destruction of the aortic root. If the valve is functionally abnormal but sinus of Valsalva disease is absent, prophylactic aortic valve replacement with retention of the remainder of the aortic root is justified. Alternatively, reparative bicuspid valve procedures are well-reported, but their application should be judicious.

The routine use of intra-operative TEE to guide and assess repair, monitor TL flow, and assess global and

regional ventricular function is now regarded as an essential adjunct (36,236,237).

Surgical Management of the Distal Aorta

The significance of a patent false lumen. A DeBakey type I ATAAD treated by proximal aortic surgery usually leaves the patient with a persistent distal patent false lumen (PFL), the importance of which has been noted for decades (38,238–241). Ostensibly, a PFL diminishes prognosis (38,102,222,242–247), being associated with an increased risk of death (248,249), re-operation (248,250), and aneurysmal enlargement (213,251–254). In some reports a PFL is associated with a 20% and 25% reduced chance of actual and event-free survival at 5 years (212,255), whereas in others a much lower effect is observed (256). Uncertainty of the effect size of PFL on longer-term outcome fuels a major debate with regard to the need for more extensive repair.

The region at most risk of aneurysm formation is the distal arch and proximal DescAo (213,248,257,258). Natural history studies consistently demonstrate a heightened expansion rate in the PFL aorta (213,259), but expansion might be slow, linear, and limited (249). Nearly 50% of PFL aortas will experience growth rates <1.0 mm/year, and notably dissection-related distal aneurysms cause only a minority of late post-ATAAD deaths (255). The number of patients requiring late elective re-operation within 5 years in most series is 2% to 13% (105,212,213,224,249,255,256,260) but higher in some (261,262). Marfan's patients are an exception; however, if these are excluded relatively few patients develop late aneurysmal enlargement of the aorta (255). Importantly, re-operations might be performed with low mortality (249). However, follow-up is often incomplete, and the potential for aorta-related mortality might be higher than the re-operation rate suggests (212). The re-operation rate after AscAoR or hemi-arch replacement might not be appreciably higher than rates reported after more extended arch replacement (263–265). The factors that might pre-dispose to aneurysm are: pre-existing aortic dilation (213,259); uncontrolled hypertension (259); non-resection of an entry tear (102,110,246,259); the presence of larger-dimension FL diameters (in some but not all reports) (247,262) and area (262,266); and as seen in type B dissection (267), partial FL thrombosis (268). In the presence of a PFL, vigilant life-long surveillance with interval scanning is necessary, preferably with magnetic resonance imaging. Pragmatically, scans at 3 and 6 months and then annually seem satisfactory with interval adjustment if accelerated enlargement or, conversely, stability at normal dimensions are detected. Repeat surgical review is advisable if a minor diameter dimension of 5.5 cm is reached or expansion rate exceeds 1 cm/year^{-1} (23). Such patients might be candidates for consideration of intervention (269).

Operative techniques and the residual PFL. CLOSED VERSUS OPEN REPAIR. The most common site of the intimal tear is the proximal half of the AscAo (13). This might allow a closed technique of AscAoR undertaken with the distal AscAo clamped throughout the procedure. The alternative is an open distal anastomosis (ODA) technique, which demands clamp removal with an anastomosis being performed during a period of hypothermic circulatory arrest (HCA) often supplemented by adjunctive brain perfusion techniques. The closed technique has certain advantages. It is quicker, because it avoids the necessity of profound cooling and prevents the introduction of additional air and debris into the aortic arch (270). It is particularly applicable to the small number of ATAAD–DeBakey type II dissections limited to the AscAo proximal to any clamp position. Closed techniques do not have worse operative mortality than open anastomoses (212,215,242,259,270–273) unless the inclusion technique that does not use full thickness suturing is used (274,275). In addition, long-term survival outcome might be similar (Online Fig. 4) (212). However, a closed technique disregards secondary tears within the aortic arch present in 20% to 30% of DeBakey type I ATAAD (13,14,276), and the friable dissected AscAo might be injured by the clamp instrument, generating further intimal tearing. A closed technique results in near-uniform PFL persistence in DeBakey I dissections (257,272). Because it facilitated more complete repair and hypothetically reduced PFL risk, ODA became both popular (277) and the accepted surgical norm (14,278). An ODA allows a more accurate approximation of the dissected layers and a direct visual inspection for further arch tears (279). If a clamp has been applied during cooling, the fragmented clamp site might be excised during ODA reconstruction, potentially limiting PFL rates. However, although lower PFL rates are reported, they remain disappointingly high (60% to 80% in DeBakey type I ATAAD) (49,212,253,254). Nevertheless, current consensus favors an ODA. It allows anastomotic reconstruction in a fully visualized aorta and offers the opportunity to provide a hemostatically secure anastomosis, to extend the repair into the hemi-arch or beyond, to attempt re-approximation of the intimal flap, and to detect and treat additional arch tears.

GLUE FIXATION OF THE DISTAL AORTA AND ANASTOMOTIC TECHNIQUES. Despite the purported utility of surgical glues (221–223,243,280) to secure adhesion between intimal flap and FL wall in the distal aorta, there is little evidence that they reduce PFL rates beyond that achieved by an adhesive-free ODA technique (49,257). Reduced re-operation rates have been observed in some series but not others (215,224,272,274), and there is concern with regard to adhesive safety (223,274,281–283). Over-exuberant glue application in the distal aorta might extrude through secondary tears, causing branch vessel occlusion or embolization, and many surgeons withhold its use in the distal aorta (284).

EXTENSION OF THE AORTIC REPLACEMENT INTO THE ARCH. Because an ODA has little effect on PFL prevalence, surgeons have attempted to increase repair extent to reduce PFL rates. When the ODA is advanced to replace the

under-surface of the proximal aortic arch (constituting an extended ODA or hemi-arch), surgical risk remains unchanged (158,212,272,279,285), but the effect on PFL rate is limited. More extensive repairs requiring total aortic arch replacement (TAR) have reduced PFL incidence to nearly 25% in some but not all reports (105,258,260,263,264,286–294), but this might be at the expense of increased early mortality risk (295), particularly in elderly patients (84). Although some report improved longer-term outcomes (287), others have not observed any reduction in late re-operation rates (261,292,296,297). It is unlikely that this more complex approach could be generalized to the whole cardiovascular surgical community and registry data could continue to demonstrate that TAR is associated with increased surgical risk (46). Modifications that might simplify TAR are reported, but longer-term outcomes are not yet formally reported (265,294).

HYBRID EXTENDED REPLACEMENT PROCEDURES: BARE-METAL STENTING AND SEPARATELY DEPLOYED COVERED STENT-GRAFTS. One approach to reduce PFL and promote dissection regression and aortic remodeling involves the deployment of a bare-metal stent that compresses the true lumen against the outer medial-adventitial wall, promoting FL thrombosis, TL expansion, and medial healing by cicatrisation. This could avoid TAR and does not affect epi-aortic artery patency. Although a PFL in the aorta beyond the stent would remain, the vulnerable distal arch and proximal DescAo (262,266) might be protected. Preliminary reports using stainless steel balloon-dilatable stents (Fig. 5) suggested that FL obliteration could be achieved in >70% of patients (298–302), but recent reports suggest little PFL rate effect and no FL size reduction (303). Incomplete deployment might occur, and if inaccurate, there might be a heightened risk of aortic rupture. Whether future developments will improve outcomes must be the subject of detailed cohort studies with contemporary matched control subjects.

An alternative approach combines hemi-arch with covered stent-graft of the upper DescAo. Depending on the extent of proximal repair, this technique might leave the middle section of the aortic arch untreated. Covered stent grafts are deployed either retrogradely or via the open aortic arch. Preliminary reports suggest a reduction in PFL and possibly late aneurysm development (150,304,305).

Stent-prosthetic graft combinations: the frozen elephant trunk technique. The elephant trunk procedure (306), in which a length of prosthetic graft beyond the distal arch anastomosis in TAR floats freely within the DescAo, facilitates interval repair of distal aortic aneurysms (307–309). A recent modification, with a covered endovascular stent-graft attached to a vascular graft, allows fixation of the stent graft within the DescAo with vascular graft reconstruction of the arch (310). This frozen elephant trunk (FET) technique has facilitated single-stage TAR and DescAo replacement in complex aneurysms and has been extended to ATAAD (Fig. 6) (151,311).

The FET technique has been demonstrated to promote FL thrombosis, reduce distal PFL rates and deal with secondary tears in the arch and re-entry tears in the proximal DescAo (149,150,311–317). Patent false lumen rates of 10% versus 89% in non-FET patients are reported. The stent graft might expand a compressed true lumen and thereby ameliorate distal aortic malperfusion. However, although FET and other more complex stent-graft combinations might be found to decrease late aneurysm formation (149,318–320), the procedures require total or substantial arch replacement, automatically extending cerebral protection time and the period of corporeal circulatory arrest. This might increase stroke and paraplegia risk (320,321,322). One certain indication for FET is ATAAD with DescAo rupture (323), and it might also have a particular role in retrograde dissection (324,325), but beyond these, indications are uncertain and controversial (326–330). Because

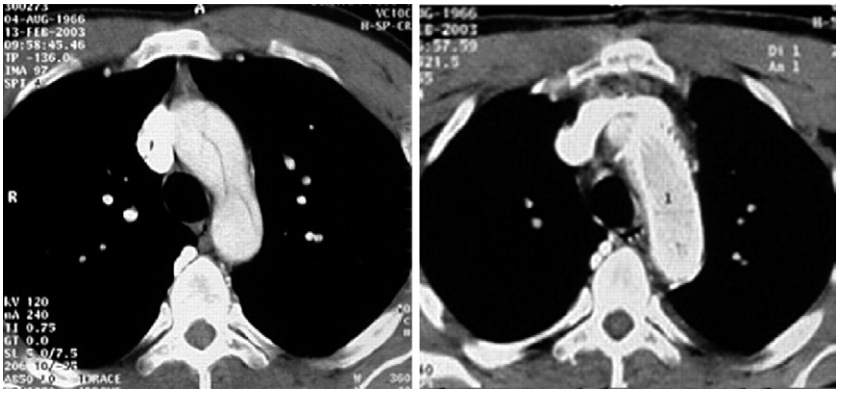

Figure 5 Computed Tomography to Demonstrate a Type A Aortic Dissection and Repair

Computed tomography to demonstrate a type A aortic dissection with extension into the aortic arch **(left)**. The right-hand post-operative image demonstrates placement of an intra-operative stent-graft after ascending aorta repair. Note the resolution of the false lumen. Reproduced, with permission, from Leobon et al. (299).

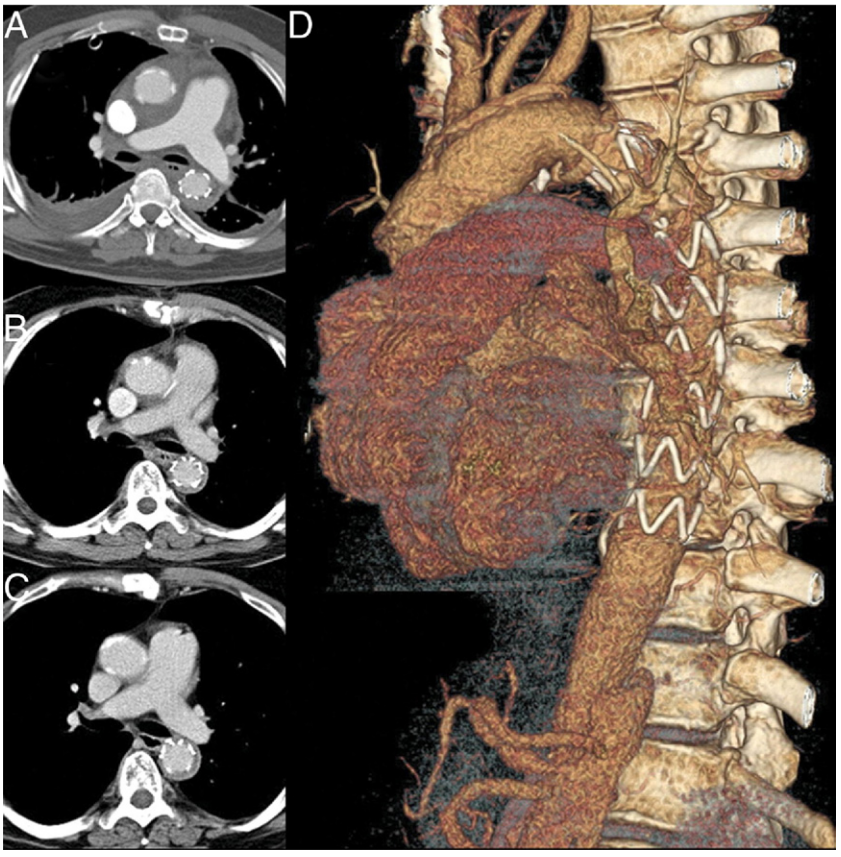

Figure 6 **Hybrid Frozen Elephant Trunk Technique for Management of the Descending Aorta**

Computed tomography scans at **(A)** 10 days, **(B, D)** 6 months, and **(C)** 12 months demonstrating continued thrombosis and exclusion of the false lumen in the descending aorta. In **D**, the false lumen is seen to refill in the distal descending aorta. Reproduced, with permission, from Jakob et al. (149).

5-year re-operation rates are considerably <15% in most series (211,225,244,250,254,317) and can be performed with modest surgical risk, patient subgroups that definitively derive long-term benefit that might justify perioperative risk need to be identified. Currently, whether patients with risk factors for accelerated life-threatening aneurysm development have a lower overall hazard with FET techniques versus the cumulative risks of more conservative surgery, surveillance monitoring (with an anticipated attrition rate), and late re-intervention is unknown. In 1 series, FET-treated patients had better medium-term survival than a conventionally treated but higher-risk control group (291). Sun et al. (331) have reported low operative mortality in patients with ATAAD undergoing ascending repair with TAR and ET. Distal FL obliteration in this group was significantly reduced, compared with those undergoing conventional repair (94.2% vs. 14.5%, respectively); this has not as yet translated into a long-term survival benefit for the more extensive procedure, compared with conventional repair in the setting of ATAAD.

Thus, despite the impressive aortic remodeling data in downstream segments, such extensive procedures remain an unproven alternative to conventional ATAAD repair. Additionally, any increase in surgical complexity and extent must recognize their adverse relationship with patient outcome.

Cerebral protection and monitoring during ATAAD repair. A full review of intra-operative brain protection in ATAAD is beyond the scope of this review. There is no randomized trial evidence in ATAAD and only a small amount in elective arch surgery (23). However, clinical consensus has been reached in some areas. Because an ODA is considered optimal practice, a period of HCA is necessary. Even profound HCA provides only limited periods of brain safety (approximately 20 min) (332), sufficient only to perform a simple ODA. The HCA is insufficient for more extensive reconstruction, and adjunctive brain protection techniques are necessary. Retrograde brain perfusion via the superior vena cava as an adjunct to deep hypothermia may not be used, but the majority of arch procedures are now undertaken with antegrade brain perfusion (333). The delivery of antegrade perfusion may be via various routes (334,335). Questions remain with regard to the ideal route, the optimal perfusate composition, and the temperature of

perfusate and patient during the corporeal arrest period. It is now recognized that corporeal arrest temperature is highly relevant to spinal cord protection (336,337). In the context of ATAAD, corporeal and cerebral protection techniques and intra-operative monitoring to detect malperfusion are essential components of the surgical strategy in striving for a satisfactory complication-free patient outcome (23).

Summary

Acute type A aortic dissection requires rapid diagnosis and treatment, if associated mortality is to be reduced. Although imaging-based diagnostic accuracy is excellent, the ultimate acquisition of the diagnosis is commonly delayed. Referral and transfer for operation incurs further delay, and surgery is commonly provided in nonspecialist units. Patient selection for medical treatment should be based upon a comprehensive appreciation of risk factors, including comorbidities and dissection-related complications and not on a single factor such as age alone. Malperfusion phenomena are common, invoke diagnostic delay, and compromise survival. In most cases, reperfusion as soon as possible after symptom onset achieves superior outcomes. In an effort to treat more patients and improve outcomes, we advocate: 1) research into pre-hospital diagnostic recognition; and 2) establishment of designated specialist units to facilitate all management.

Within surgical therapy, there are a host of controversial issues that require resolution if outcomes are to be improved. The evidence to determine best surgical management is unlikely to be drawn from individual center studies reporting surgeon-specific techniques in limited numbers, comparative retrospective reports with historical controls, or prospective reports in which more novel therapies are directed toward more stable patients with consequent apparent advantages. Randomized controlled trials of surgical technique seem extraordinarily difficult for several reasons. These include the difficulty in obtaining informed consent in an emergency situation, the heterogeneity of the patient population and pathology, and funding in the absence of novel pharmacological agents. We believe that the most efficacious way to address these many controversies is by the accrual of registry data. Such registries should be large and multinational, accruing very detailed perioperative data with standardized reporting definitions. The detail is required to clearly define the patient, clinical status, pathology, pathological anatomy, and surgical findings and details of the anesthetic, monitoring, and surgical techniques. Imaging, both at diagnosis and follow-up, should be subjected to core laboratory review and reporting, and outcome data should be assessed independently. In this way, we will, in due course, be able provide the evidence that informs surgical practice and optimizes the management of this highly dangerous condition.

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Key Words: evidence ■ surgery ■ treatment ■ type A aortic dissection.

APPENDIX

For supplementary figures and tables, please see the online version of this article.