The issue

Surgical outcomes after repair of acute type A aortic dissection have steadily improved during the last decade. The main reasons for that have been the switch to cannulating the right subclavian artery for arterial return, the increased use of selective antegrade cerebral perfusion while using hypothermic circulatory arrest at warmer temperatures, the broader use of cerebral monitoring thereby decreasing the incidence of symptomatic central neurologic injury and finally the better understanding of the underlying disease with regard to the location of the primary entry tear and the associated presence or absence of malperfusion and its treatment (1-4). However, a substantial number of patients having successfully undergone primary repair will return with the need for secondary repair because of aneurysmal formation in primarily non-repaired segments (5). Some of them are fateful but many are predictable already at the time when interpreting the referral CT scan or the initial completion CT scan.

Underlying mechanisms and extent of disease as well as extent of repair

Closure of the primary entry tear remains the basic principle in any kind of acute and chronic dissective aortic pathology irrespective of segment and extent (4,6,7). The location of the primary entry tear finally determines the strategy. In a scenario where the primary entry tear is located in the mid-ascending aorta, ascending aortic and very important hemiarch replacement will fix the entire disease—presupposed that diameters in downstream segments are regular. In a scenario where the primary entry tear is located in the distal aortic arch or even in the proximal descending aorta—a scenario which is often associated with visceral and/or renal malperfusion—ascending and hemiarch replacement may prevent rupture but will leave the underlying disease mechanism unaddressed thereby often leading to rapid diameter expansion. This mechanism has been described in a study investigating similarities and differences in patients with primary type B aortic dissection...
and patients with chronic type B residual after type A repair where a remaining patent primary entry tear (or a large communication between lumina) independently predicted the need for secondary intervention [surgery or thoracic endovascular aortic repair (TEVAR)] (8).

However, even in patients where the primary entry tear has been closed during primary surgery, secondary intimal ruptures at the arch anastomosis and either multiple or large communications between lumina across the membrane can function as new primary entry tears thereby also leading to diameter enlargement warranting treatment sooner or later (9).

It is the authors’ experience that short ascending grafts leaving a substantial amount of native tissue in place as well as the affection of supra aortic branches are also associated with a higher probability of developing late post-dissection aneurysmal formation whereas correct hemiarch replacement in the absence of initial large diameter in downstream segments and without large communication between lumina (i.e., below 40 mm total aortic diameter) will have a high probability of a stable course during follow-up.

**Successful and unsuccessful treatment approaches and options**

A decade ago, combined vascular and endovascular approaches have gained popularity in patients with proximal thoracic aortic pathology in order to create a sufficient proximal landing zone for secondary TEVAR (10). This approach was very successful in descending pathologies originating at the level of the aortic arch by subclavian-to-carotid transposition or bypass and also double-transposition became widely applied technique whereas total aortic arch rerouting was associated with a high incidence of retrograde type A aortic dissection when the proximal anastomosis was done to a native ascending aorta (11). In prosthetically replaced ascending aortas—like it is the case after repair for acute type A aortic dissection, this complication by nature could not occur and several groups used the approach for treating chronic type B residual after previous type A repair. The concept worked; however, several limitations became obvious (12). As mentioned, one of the reasons why aneurysms develop in this scenario is that ascending grafts are left too short which also limits the possibility to create a sufficient landing zone within the graft. Additionally, affection of the supra aortic branches was an issue as under beating heart conditions lumen identification and thereby correct accomplishment of vascular anastomoses was challenging. Finally, exposure of the left subclavian artery was demanding in cases where the distance between the sternum and the native left subclavian artery was more than 10 cm.

Additionally, the success of TEVAR was dependent onto the size of the true lumen which might be very small in particular in cases where the primary entry tear has not been closed during primary repair. Additionally, indwelling stent-grafts in small true lumina may cause distal stent-graft induced new entries (dSINEs) by a mismatch between the often (but not always) rigid chronic dissection membranes sooner or later (13). These dSINEs functionally act as new primary entry tears thereby shifting the initial mechanism more distally. Finally, success was most pronounced in cases, where there were no or very few communication between the lumina, all visceral and renal offsprings where from the true lumen and the punctum maximum of aneurysmal formation was at the level of the distal arch and/or the proximal descending aorta respectively. After all, the time between the initial acute event and the need for secondary intervention had a clear correlation with the potential of downstream aortic segments to remodel as this effect is most pronounced within the first 2 years after the acute event.

With the advent of the frozen elephant trunk (FET) technique, many patients with chronic type B residual after previous type A repair underwent FET implantation which nicely addressing the entire native thoracic aortic pathology from the level of the indwelling previous ascending repair including the entire arch and the offspring of the supra aortic branches up to the level of the descending aorta where the stent-graft component of the FET prosthesis achieved the same effect as primary TEVAR in primary type B scenarios or in the same scenario using a combined vascular and endovascular approach (14-16). In patients where the aortic root was either normal or already addressed by valve sparing or classical aortic root replacement, a beating heart technique is increasingly used where we do use in our setting a modified Hannover protocol with normothermic blood being infused to the aortic root proximal to an aortic clamp and the entire arch replacement in performed in lower body hypothermic circulatory arrest and selective antegrade brain perfusion with an emptied beating heart and a normal electrocardiogram (17) (Figure 1). This approach in our experience seems to reduce the need for vasopressors and inotropes when weaning from cardiopulmonary bypass and thereafter to a minimum. Figure 1 shows a FET implantation using the Thoraflex™ Hybrid prosthesis (Vascutek, Scotland) in a case of chronic
type B residual after previous type A repair. The details of our conceptual approach using this method have been described previously (19).

However, the limitations as could be expected remained similar being dSINE as well as continuing growth in downstream segments despite successful primary entry tear closure. Also when using the FET technique, the effect was highly dependent onto the location of the maximum diameter, the time interval between the acute event and the FET procedure and finally from true and false lumen offspring of visceral and renals with less efficacy when many communications between lumina were detectable and when major vessel offspring from the false lumen—due to increased cross flow and pressurization and thereby reduced capability to fully depressurize the false lumen was present. Initial attempts to close the false lumen, e.g., at the level of the thoracoabdominal (TA) transition have shown effect but midterm results have to be awaited before a recommendation of such a kind of approach can be provided (20).

Distal TEVAR extension may well stabilize the thoracic component of the disease but a substantial number of patients will remain in need of downstream classical surgical replacement. However, a Crawford type II TA scenario has then been converted into a Crawford type IV scenario presupposed that there is no type I/III endo leakage and that there are no major inaccessible segmental arteries within the upstream aneurysmal sac. Finally, diameter correction between large stent-grafts and classical surgical prostheses can be done with a so-called sewing collar solution meaning that the sewing collar of a vascular prosthesis is used to correct for diameter difference which can be very challenging when aiming for correction with a standard Dacron prosthesis (21). Finally, classical surgery remains an option in a chronic type B residual after previous type A repair whereas there is a fundamental difference according to the extent of initial surgery. In patients where merely the ascending aorta has been replaced, the complete and adequate fix of the pathology is total aortic arch and downstream segment replacement according to the individual extent from a posterolateral approach in hypothermic circulatory arrest which is a big operation with a high potential for collateral damage and should be reserved for the very few scenarios without the option of a two stage repair starting with arch replacement using the FET or in case if anticipated secondary surgical repair classical ET repair and then secondary TA replacement is not possible such it is the case in acute scenarios. Figure 2 shows type II TA replacement in a patient having had total arch replacement using a classical ET.

In patients with a FET or a classical ET in place, secondary classical surgery from a posterolateral approach is a safe and highly efficient operation provided that the general condition of the patient permits major surgery. In the authors’ experience, in the majority of patients presenting, the aortic pathology as their limiting disease and are rarely affected by other severe cardiovascular conditions such as heart failure, coronary artery disease or extensive valve pathology. Just for the completeness of mentioning options, a stent-graft can also be inserted into a classical ET presupposed the classical ET provides adequate length.

**Future efforts**

As technology advances, total endovascular solutions for
several proximal thoracic aortic disease processes have become available. Currently, two companies provide prostheses having been designed for orthotopic endovascular aortic arch repair (23,24). Initial results of both prostheses are encouraging and it is to be expected that technology will improve and application will be broader in the years to come. It is important to realize that these prostheses do require an adequate (i.e., 6.5 cm) proximal (prosthetic) landing zone and many patients do not qualify for the technique due to the before mentioned short ascending aortic grafts which per se also contribute to post dissection aneurysm formation. Finally, use of the technique in case of affection of the supra aortic branches by the dissection process is currently discouraged.

Summary

The incidence of patients with chronic type B residual after previous type A repair in need of treatment is not trivial and several parameters indicate a high probability already at the time of primary repair permitting prevention by an individualized strategy at initial surgery mainly aiming at the closure of the primary entry and of large communications between lumina either by either extending classical repair or by using the FET technique. The FET technique—and in case of suitability—combined vascular and endovascular approaches serve as highly efficient means to address the remaining native arch and proximal descending aortic segments. However, tertiary endovascular and eventually classical surgical distal repair is frequently needed for a full fix. Continuing surveillance and an anticipative strategy remains the mainstay in patients with chronic type B residual after previous type A repair.

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Footnote

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References


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