The surgical repair of thoraco-abdominal aortic aneurysms (TAAAs): state of the art

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Kardiochirurgia i Torakochirurgia Polska 2005; 2 (4): 14–17

The surgical repair of TAAAs has always been and still is the ultimate challenge of cardiothoracic and major vascular surgery. From the anatomical standpoint an aortic aneurysm can be identified as "thoraco-abdominal" if both the visceral segment and the thoracic aorta are involved in various degrees. According to Crawford's classification, TAAAs can be divided into four different extents based on their extension along the aorta (Fig. 1).

Despite the finest Medical Institutions around the world working hard to reduce morbidity and mortality associated with TAAA repair over the past 50 years, intraoperative and postoperative complications such as paraplegia, paraparesis, acute renal failure (ARF) and acute respiratory distress syndrome (ARDS) may still occur with significant incidence (0-40% for paraplegia and 5-20% for ARF). These complications are severe since the lesions are usually irreversible and might also significantly influence patient's survival.

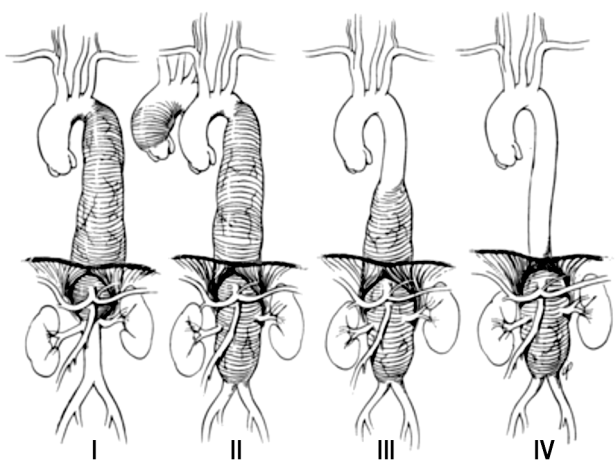

Fig. 1. Crawford' classification of thoracoabdominal aortic aneurysms. **Extent I**, distal to the left subclavian artery to above the renal arteries. **Extent II**, distal to the left subclavian artery to below the renal arteries; this is the most extensive type of aneurysm and the only extent that is still associated with risk of paraplegia, with the use of adjunct distal aortic perfusion and cerebrospinal fluid drainage. **Extent III**, from the sixth intercostals space to below the renal arteries. **Extent IV**, from the twelfth intercostals space to the iliac bifurcation (total abdominal aortic aneurysm)

During the last half century several methods of intraoperative protection of the visceral organs and spinal cord have been proposed and eventually used as common practice. Other methods regarding complications to either general cardio-thoracic surgery or major vascular surgery have also been proposed in order to reduce the incidence of adverse events such as respiratory and cardiac failure, postoperative bleeding and thromboembolic events.

Nevertheless, the most severe complication of TAAA repair still remains ***postoperative paraplegia*** and this explains why most research around the world over the last five decades has been targeting this issue. During the early 1950s Michael DeBackey et al. tried to use cold water infusion through the intercostal arteries in the attempt to reduce spinal cord ischemia (spinoplegia). Subsequently Hollier et al. developed the CSF-drainage technique, eventually modified by Svensson et al. with the use of papaverin infusion into the CSF-catheter and by Acher et al. with Naloxone infusion. Dr. Stanley Crawford in Houston first proposed the "clamp and go" technique which was based only on the surgeon's ability to "sew fast", regardless of any hypothermic organ protection. More recently Cambria et al. have proposed "epidural cooling" which is a spinoplegia performed by cooling down the epidural space. Among all these methods, only the CSF-drainage has been proven effective in reducing intraoperative and postoperative spinal cord damage.

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Another technique of organ protection developed over the years involves the continuous perfusion of visceral organs along with sequential (proximal-to-distal) cross-clamping of the various segments of the aorta, achieved by extracorporeal circulation. The original technique used during TAAA repair was a femoro-femoral by-pass. This technique was associated with high incidence of postoperative bleeding due to the effect of complete eparinization (3 mg/kg) on TAAA-patients who usually experience poor bleeding control because of wide surgical incision (thoraco-phreno-laparatomy) and complex surgical procedure such as the replacement of the entire thoraco-abdominal aorta. Dr. Denton Cooley first proposed the use of a left-heart by-pass achieved by a "Bio-Pump" which involves the use of a cannula in the left atrium and another cannula in the distal aorta or in the femoral artery and takes the arterial blood from the upper to the lower segments of the aorta during sequential cross-clamping. This technique does not have the drawbacks of CPB because it requires only an incomplete eparinization (1mg/kg) and has been proven very effective in reducing the incidence of paraplegia during TAAA repair.

At the present time, only Kouchoukos et al. are using deep hypothermia and circulatory arrest (DICA) in the surgical treatment of standard TAAAs with very low incidence of postoperative paraplegia (2-6%), but high incidence of complications related to DICA itself such as respiratory distress and coagulation disturbances. All remaining Authors are using regular left-heart by-pass for standard TAAA repair, reserving DICA for patients with TAAAs involving the aortic arch, when proximal cross-clamping cannot be achieved with adequate residual brain perfusion through the anonymous trunk and the left common carotid artery.

In conclusion, in our current practice spinal cord protection during TAAA repair is performed as follows: Ext I-II-III TAAA repair, according to Crawford's classification, involves the use of left-heart by-pass and CSF- drainage; Ext IV TAAA repair involves the use of the "clamp and go" technique, without additional organ protection.

It is widely accepted that reimplanting intercostal arteries is essential in order to prevent postoperative paraplegia after TAAA repair. The spinal cord has a very complex and fragile vascularization. The scheme drawn below shows the mainstreams of blood supply to the spinal cord. The top portion of the spinal cord, including the segment giving off the nerves of the cervical plexus, is supplied by the vertebral artery of both sides (arising from the subclavian artery) which gives off branches then joining together into the left anterior spinal artery. All pairs of posterior intercostal arteries arising from the descending aorta give off collaterals to the anterior spinal artery. The contribution to spinal cord supply of each one of those has a significant variability in the general population. It is widely accepted, however, to divide intercostal arteries in upper Ias (between T1 and T7) and lower Ias (between T8-T12). Everybody agrees that lower intercostal arteries are critical to the perfusion of the anterior horns, giving off the nerves of the lumbar plexus. Among these pairs should be identified the so-called "Adamkiewicz artery", a pair of low posterior intercostal arteries or high lumbar arteries especially important for spinal cord supply. There is no evidence, however, that such a pair could be identified with statistical significance in the general population.

The lumbar arteries are in series with the posterior intercostal arteries and there are usually 4 on each side. They give off a dorsal ramus (supplying the muscles of the back) which also furnishes a spinal branch supplying the "cauda equina". The lumbar arteries anastomose with lumbar branches of the iliolumbar artery (branch of the internal iliac artery). It ascends between the vertebral column and the psoas major muscle and divides into a lumbar and an iliac branch. The lumbar branch goes to the psoas and the quadratum lomborum muscles but it also sends a spinal branch to supply the "cauda equina" which also anastomose with lumbar arteries.

There is a lot of controversy on how and when intercostal arteries should be reattached. Our current strategy is the following: at the opening of the native aorta the pairs of posterior intercostal arteries might be either occluded or open. If occluded, we have to assume that a collateral circulation has already been developed. If open, the intercostals can either overbleed (good run-off) or underbleed or not bleed at all (poor run-off). The rationale is to sew up the bleeding ones and reimplant the underspilling and those not bleeding at all. The reimplanted intercostal should be rounded up in a single patch of native aortic tissue as smaller as possible. This technique might be difficult in the case of calcified or dissected aortic tissue. We do not use cold blood or crystalloid perfusion of reimplanted intercostals in the ischemic time. As reported by many Authors, intraoperative "evoked potentials" might be used in the assessment of postoperative spinal cord damage, but we do not use them routinely. Appropriate reattachment of intercostal arteries and careful monitoring of the CSF-pressure (to be kept < 10 H20 cm) are the best tools to protect spinal cord from either intraoperative or residual ischemia.

Despite all these methods the risk of postoperative paraplegia in TAAA ext I, II, III repair cannot be eliminated. In a multiple regression analysis the following have been shown to be independent predictors of postoperative paraplegia: patient's age (especially if over 70), the extent of the lesion (the risk is significantly higher in ext II TAAAs),

the type of lesion (the risk is significantly higher in dissecting aortic aneurysms), aortic cross-clamping time and the type of onset (elective or emergent). Intraoperative hypotension should be carefully avoided and the intraoperative anesthesiologic management of the TAAA patient is critical to surgical outcome. Late-onset paraplegia might occur between 48-72 hours after surgery and is usually secondary to either angled or thrombosed reimplanted intercostals.

Another significant issue in terms of postoperative complications of TAAA repair is ***postoperative acute renal failure*** (ARF). A certain level of renal dysfunction is very common in the first 48 to 72 hours after TAAA repair and is usually reversible. Only when creatinine levels exceed 3 mg/dl a true ARF is present. Clinical features are variable. In patients with preserved urinary flow usually creatinine levels go back to normal under adequate drug regimen. On the other hand, anuric patients should be treated aggressively with early ultrafiltration (CVVH). This condition may evolve either toward spontaneous resolution with a polyuric phase or toward the need of permanent dialysis.

Intraoperative cold crystalloid renal perfusion (at 4°C, according to Coselli's technique) is a valuable tool to prevent postoperative ARF. We do not use it on a regular basis but only on single-kidney patients (which is not uncommon in dissecting aortic aneurysms) and in patients whose preoperative creatinine levels exceed 2-2.5 mg/dl. After reimplanting the visceral patch, we inject Indocianin blue into the general circulation. The rationale is either to test renal clearance (if urine does not colour within 30 minutes we check up on both renal artery anastomoses to rule out angles or intimal flaps) or to test ureter integrity in cases where the surgical fields colour (which is especially important in redo operations where anatomy is disrupted and the ureter can be virtually everywhere).

In a multiple regression analysis the same risk factors of postoperative paraplegia have been shown to be independent predictors of postoperative ARF. An extra independent and very powerful predictor of postoperative ARF is preoperative chronic renal failure (CRF).

Postoperative bowel necrosis is another rare but devastating (90% mortality) complication of TAAA repair, with an incidence, in our experience, of 2.5%, which is compatible to the 4% reported by most Authors. Again, we do not routinely use warm blood perfusion of the celiac trunk and the superior mesenteric artery during visceral patch reattachment, as reported by Coselli et al., since we think that in a wide majority of cases bowel necrosis is not secondary to intraoperative ischemia but to thromboembolic events. It might happen that either a portion of the atherosclerotic plaque might embolize after declamping of the aorta or thrombotic material coming from the prosthesis itself because of poor washing out might take the route of the superior mesenteric artery. This may happen quickly, or later in the recovery period (late-onset bowel necrosis). In the latter case the onset is often insidious and usually takes to a late diagnosis and the occurrence of a life-threatening situation since early and emergent surgical bowel resection, when possible, is the only chance of survival.

Postoperative acute respiratory failure is the complication occurring with the highest incidence after TAAA repair (35% to 38% as reported). Patients over 60 years of age, smokers, patients with chronic obstructive pulmonary disease (COPD) are the categories at highest risk of developing postoperative respiratory failure. The surgical incision itself, which involves the chest wall and the diaphragm, is very invasive and disruptive to the main respiratory muscles. Pain also significantly influences the depth of inspiration and the coughing mechanism which becomes ineffective in pulling out secretions. The circumferential incision of the diaphragm, sparing the phrenic nerve, only partially protects the integrity of the muscle and sometimes, in seriously compromised patients, we try to spare as much diaphragm as possible if compatible with the surgical exposition. In the recovery period patients are usually extubated within 24-48 hours and we use bronchoscopy for aggressive cleaning of the respiratory tract. In the case of prolonged intubation we perform routine tracheostomy on the 7th postoperative day. Of course, postoperative tracheostomy has a negative prognostic factor on the following recovery period since it means that the patient cannot breathe spontaneously. Other types of respiratory failure common after TAAA repair are ARDS, increased dead space, atelectasis, pneumonia and pleural effusion.

Some complications of general cardio-thoracic surgery might involve TAAA repair operations. ***Perioperative acute myocardial infarction*** (AMI) might occur in the case of underlying asymptomatic coronary artery disease (CAD). Elective coronary angiography must be part of the preoperative assessment of TAAA patients. Intraoperative AMI is a common cause of intraoperative hypotension or cardiocirculatory arrest. In the case of CAD either PTCA or surgical myocardial revascularization should be performed before TAAA repair. ***Postoperative bleeding*** is not very common, but it might become significant in the case of preexisting coagulopathy, disseminated intravascular coagulopathy (DIC), or weakness of the aortic wall such as a dissected aorta or a Marfan tissue.

As a last note, a couple of technical remarks. Under normal circumstances the surgical incision involves the opening of the peritoneal cavity in order either to have a wider surgical field or to check up on visceral vessels

pulse after reattachment. These maneuvers would be impossible via an extraperitoneal access. Only in redo-redo operations might we decide to choose the extraperitoneal technique by dissecting in between the peritoneum and the abdominal wall in order to avoid adherences. Another issue involving the proximal anastomosis on the distal arch concerns the recurrent laryngeal nerve, which arises from the vagus nerve crossing over the anterior face of the arch, turning back to the neck making a loop around the arch, next to the ductus arteriosus. This area in between the arch and the pulmonary artery must be cleared for aortic cross-clamping. Care should be taken not to damage the recurrent laryngeal nerve while dissecting in that area or while sewing the proximal anastomosis in order to avoid postoperative left vocal cord paralysis.

In conclusion, TAAA repair is an effective and successful procedure in treating life threatening situations such as thoraco-abdominal aortic aneurysms. Organ protection along with the surgeon's ability is critical to a positive outcome. Postoperative paraplegia remains the most serious postoperative complication of TAAA repair and all the efforts of research Centers around the world should be targeting this issue in the near future.