CorSalud 2014 Apr-Jun;6(2):129-139

CorSalud

Cuban Society of Cardiology

Editorial

Cardiocentro Ernesto Che Guevara Santa Clara Cuba

Surgical treatment of aortic dissection

Tratamiento quirúrgico de los aneurismas disecantes de aorta

Alberto Juffé Stein✉, MD

Head of the Department of Cardiac Surgery. Hospital Modelo. A Coruña, Spain.
Permanent Academician of the Chair of Cardiac Surgery of the Royal Academy of Medicine and Surgery of Galicia, Spain.

Este artículo también está disponible en español

ARTICLE INFORMATION

Key words: Aorta, Dissecting aortic aneurysm, Aortic dissection, Surgery, Treatment
Palabras clave: Aorta, Aneurisma disecante de la aorta, Disección aórtica, Cirugía, Tratamiento

Aortic dissection is associated with high morbidity and mortality rates. Mortality is 1-2 % per hour in the first 24 hours after the onset of symptoms; and, without surgery, 90% of patients die within the first 3 months. Patients who receive a proper emergency treatment show a hospital mortality rate ranging from 15 to 30 % [1-3].

Aortic dissection occurs when a tear in the intima causes blood to enter the media layer, forcing the layers apart. The space filling with blood between the dissected layers is the false lumen, and the pressure generated in it can lead to multiple sites where the blood flow goes in and out. In general, it may extend to the iliac arteries and affect on its way the renal and mesenteric arteries, with its consequent ischemic damage.

✉ *A Juffé Stein*
Calle del Virrey Ossorio, 30
15001 A Coruña, España.
E-mail address: albertojuffe@hotmail.com

In order of frequency, the vessels involved in type A aortic dissection are: renal, iliac, mesenteric, cerebral, coronary and anterior spinal arteries[4-7].

Aortic dissections may be classified according to their extent:

a) Classification proposed by Michael DeBakey in 1965[8]
   - Type I: The tear originates in the intima of the ascending aorta and propagates through the descending thoracic and abdominal aorta.
   - Type II: It originates in and is confined to the ascending aorta.
   - Type III: Dissection may be confined to the descending thoracic aorta (type IIIa) or may spread to the abdominal aorta (type IIIb). The dissection may also extend proximally, affecting the aortic arch and the ascending aorta.

b) Classification of Stanford, published by Daily in 1970[9]
   - Type A: It includes the dissections involving the ascending aorta, regardless of their site of origin and extent. It corresponds to DeBakey Type I and II.

- Type B: It includes dissections involving the descending aorta. It corresponds to DeBakey type III.

b) Atypical forms of dissection (acute aortic syndrome) are the intramural hematoma and penetrating atherosclerotic ulcer[9].

Surgery is indicated in intramural hematoma of the ascending aorta. When it occurs on the descending aorta, it is treated medically. Surgery is indicated when the hematoma is recurrent, the patient has a persistent chest pain or when there is an increase in the diameter of the hematoma[10].

Complete resolution of a hematoma on the descending aorta occurs in 50-80 % of all cases[11].

Penetrating ulcer of the ascending aorta is rare, but, if present, it requires surgical treatment. On the descending aorta, it requires medical treatment, with a close monitoring.

The dissection of the aorta is a catastrophic event, which is found in 0.2-0.5 % of autopsies, with an incidence ranging from 5 to 10 per million inhabitants a year[12,13].

Recently published data show that the incidence and prevalence of thoracic aneurysms, including dissections, has increased three times in the last 10 years[14].

Anagnostopoulus[15] lists the most common conditions that can damage the media layer of the aorta. These include congenital anomalies, idiopathic medial degeneration, hypertension, pregnancy, atherosclerosis, inflammatory diseases, trauma, and patients with previous cardiac surgery, among others.

Aortic dissection may start with the rupture of the *vasa vasorum* of the media layer, resulting in an intramural hematoma, with no intimal tear. Then, local bleeding may tear the intima, causing dissection[16].

Valvular insufficiency is a frequent element that accompanies proximal aortic dissection. It is present in 50-66 % of cases. Four production mechanisms have been identified:

1. The dissection dilates the aortic annulus preventing a proper coaptation of the leaflets in diastole.
2. When the dissection is asymmetric and the pressure of the dissecting hematoma depresses a leaflet below the coaptation line and produces incompetence.
3. When the annular support or the leaflets themselves are torn.
4. The intimal flap prolapses into the left ventricular outflow tract.

Bicuspid aortic valve is associated to aneurysm and dissection[17]; the ascending aorta shows fragmentation of elastin and loss of muscle cells, an alteration similar to Marfan syndrome[18,19].

CLINICAL PRESENTATION

The patient's clinical history and a good physical examination lead to the initial diagnosis: hypertensive patients, poorly controlled, sudden onset of precordial pain and a family history of aortic dissection.

Pain is present in 90 % of cases; its characteristics depend on the location and extent of the dissection. A sharp or stabbing precordial chest pain that migrates to the back or vertebral-interscapular region indicates a Stanford type A aortic dissection. In type B aortic dissection, the pain starts in the back and moves forward and to the abdomen, or to both places. It is sometimes associated with pain in the lower limbs, caused by ischemia.

The differential diagnosis includes acute myocardial infarction, acute coronary syndromes with or without ST elevation, musculoskeletal pain, pericarditis, mediastinal tumors, pleuritis, pulmonary embolism, acute cholecystitis, among others.

A history of hypertension is present in 80% of patients with aortic dissection. The dissection may be associated with shock symptoms, poor peripheral perfusion, pallor with hypotension, denoting a rupture from the aorta into the pericardium, with signs of cardiac tamponade. Sometimes, aortic regurgitation is present, with or without coronary dissection, and decreased or absent distal pulses (partial and complete obstruction of the subclavian or femoral arteries).

The patient may have neurological impairment including syncope, transient or permanent stroke, paraparesis or paraplegia due to spinal cord involvement; its cause is the poor perfusion of the branches of the aortic arch or the medullary arteries.

Less frequent symptoms include superior vena cava occlusion, dysphonia, compression of the tracheobronchial tree, and hemoptysis or hematemesis.

The diagnosis of aortic dissection is sometimes complex. Chest radiography, echocardiography (ECHO) and computed tomography (CT) are the tests of choice. Angiography and magnetic resonance imaging may be

useful.

In some hospitals, a CT scan is preferred for its speed, sometimes diagnosis is reached in 20 minutes; transthoracic echo (TTE) or transesophageal echo may take longer, especially if the patient is admitted at night. The TTE is useful to rule out cardiac tamponade [20].

SURGICAL MANAGEMENT

Although some patients die suddenly, most of them get to a hospital which allows diagnosis, stabilization and treatment. Surgical results depend on the severity and extent of the dissection, the age and clinical status of the patient, and the experience of the medical-surgical team.

Broadly, the dissection of the ascending aorta requires surgery, in order to prevent the progression of the dissection and intrapericardial rupture. If the aortic dissection causes poor perfusion of the supra-aortic trunks, spinal cord, abdominal viscera, kidney or lower extremities, it does not preclude early surgical treatment. Thus the dissection is solved and then, if needed, malperfusion is treated.

Patients with significant brain damage, visceral gangrene, or irreversible shock are a contraindication to surgery.

Surgical treatment of aortic dissection on the descending aorta is still controversial. In those cases with thoracoabdominal aortic rupture emergency surgery is indicated. Medical treatment is the treatment of choice to stabilize the patient.

Mortality in type B aortic dissection is 21-67 % with medical treatment and 30-40 % with surgical treatment[21-25]. However, when complications arise, endovascular or surgical treatment is the treatment of choice. Jonker *et al*[26] analyzed 583 patients with complicated aortic dissection, divided into 3 groups, with patients over 70 and less than 70 years of age. The results showed a hospital mortality rate of 10.1 vs. 30 % in the group with endovascular treatment (over 70 and less than 70 years) p=0.001; 17.2 vs. 34.2 % in the surgical group (p=0.0027) and 14.2 vs. 32.2% in the medical treatment group (p=0.001). Age was a predictor of mortality in the multivariate analysis and there was no significant difference in hospital mortality between the three groups.

Generally, endovascular therapy is indicated in urgent cases, because it is less aggressive compared with surgical treatment, especially in patients over 70 years of age with comorbidities.

PREVENTION

Reducing risk factors in patients with aortic dissection is vital (blood pressure, high cholesterol, obesity, diabetes and smoking).

Beta blockers have been used in patients with Marfan syndrome, to decrease stress in the aortic wall and prevent the growth of the aneurysm, because of its negative chronotropic effect. This therapy has also been indicated in patients with dilated ascending aorta and bicuspid aortic valve[27].

Physical exercise should be limited in patients with aneurysms larger than 4.0 cm[28-30].

MEDICAL TREATMENT (MT) OF THE DISSECTION

The primary objective is to reduce the force of contraction of the left ventricle in order to reduce aortic pulse wave (dP/dt) and lower systolic blood pressure (BP), between 100 and 120 mmHg, without compromising the perfusion of vital organs. Analgesia is one of the mainstays of treatment. The pain increases BP and the possibility of progression of the aortic dissection.

The drugs of choice are beta blockers. It is advisable to slow the heart rate below 60 beats per minute with propranolol (1-10 mg bolus and 3 mg/hour), labetalol in 20 mg bolus followed by 20 to 80 mg every 10 min, with a total dose of 300 mg or infusion (0.5-2.0 mg/min). Calcium antagonists have been used, such as the sublingual nifedipine for refractory hypertension, together with intravenous verapamil or diltiazem.

If a rapid decrease in BP is necessary, sodium nitroprusside may be used (infusion of 0.25-0.5 mcg/kg/min) depending on the response. Nitroprusside may increase dP/dt, so it should always be used together with beta blockers.

If the patient shows severe hypotension, it is probably due to cardiac tamponade, aortic regurgitation or impaired left ventricular function.

CEREBRAL PROTECTION DURING AORTIC SURGERY

Protecting the brain against hypoxic or ischemic damage is one of the challenges faced during surgery of the aortic arch and supra-aortic trunks. Surgical operations on this area require the performance of surgical

techniques to prevent or minimize damage.

The first successful replacement of the aortic arch was conducted by DeBakey in 1957[8]. These first procedures were performed with the help of temporary grafts from the ascending aorta to the brachiocephalic trunks. Subsequently, cardiopulmonary bypass (CPB) made possible antegrade flow through these trunks. In 1975, Griepp popularized the use of deep hypothermic circulatory arrest, which helped improve the results of the technique. However, in cases of circulatory arrest for more than 60 minutes, the incidence of brain damage is more likely.

Hypothermia and ischemia

The protective effect of hypothermia in ischemic conditions seems to be proven. The lower the cerebral metabolism, the greater the tolerance to ischemia; which results in a hypothetical "safety" curve during the period of ischemia. However, there will always be metabolism no matter how much we lower the temperature; therefore, the duration of a "safe circulatory arrest" is not infinite despite hypothermia.

Techniques of cerebral protection in aortic arch surgery

The main problem of aortic arch surgery with deep hypothermic circulatory arrest is that it increases the likelihood of side effects: coagulopathy, postoperative bleeding, pulmonary insufficiency, infection, among others. Deep hypothermia is less well tolerated than moderate hypothermia, especially with increasing age. To avoid these drawbacks, numerous surgical methods that optimize cerebral protection have been developed.

SELECTIVE CEREBRAL PERFUSION

It is the most logical method and the first to be developed. The brain is perfused by its usual feeding arteries while the aortic arch is sutured. Arriving at 22 °C, after cooling on CPB, the flow stops, then the three arch vessels are clamped and the aorta is opened. Thereafter, perfusion starts via the innominate trunk and the left carotid artery through flexible silicone cannulas 14 F. A flow of 10 ml/kg/min (500-900 ml/min) is kept, which is enough for a right radial blood pressure of 50-70 mmHg.

Advantages

In principle, it allows unlimited time for decision-making and proper surgical technique. It allows the use of open anastomosis (without aortic clamping).

Disadvantages

a) Need for aortic arch dissection, cannulation and clamping of trunks with danger of embolization.
b) Specific CPB equipment.
c) Operative field with too many cannulas, causing the potential hazard of a bend and ultimately, the surgeon's discomfort.
d) Lack of information on the exact flow that provides adequate cerebral protection.

Another possibility would be a moderate hypothermia at 25-26 °C and antegrade cerebral perfusion, postulated by Comas *et al*[31].

In recent years, the use of cannulation of the right axillary artery[32-34] has been proposed to carry out CPB and perform antegrade arterial perfusion during cooling and warming. By infusing the blood by a single supra-aortic trunk, unlike the above technique, the patency of the Circle of Willis is required for an adequate brain perfusion.

RETROGRADE CEREBRAL PERFUSION[35,36]

It is less and less used. It irrigates the cerebral bed perfusing the superior vena cava in a retrograde intracerebral way, draining after that into the carotid arteries.

Advantages

It is a simple and rapid method. It gives the possibility of open anastomosis, without dissecting the aortic arch, and allows drainage of possible microemboli in cerebral circulation.

Disadvantages

The time limit of the circulatory arrest remains uncertain and is shorter than in antegrade cerebral perfusion. The ideal perfusion pressure is unknown, although it is usually implemented between 15-25 mmHg. A disproportionate increase in pressure can lead to cerebral edema.

Spinal cord protection during aortic surgery

Due to the anatomical peculiarities of the spinal cord arterial vascularization, its ischemic injury is the most feared complication of aortic surgery, especially when operating on its thoracic-abdominal portion. In recent years, the rate of paraplegia associated with surgery of the thoraco-abdominal aorta has been declining, for two main reasons[37-41]: first, there are better diagnostic methods and the risk of spinal cord ischemia is better defined, and secondly, the incorporation of new methods for prevention of this type of ischemia.

However, it remains a clear risk ranging from 0.2 to 20%. One of the key determinants is the peculiar vascular anatomy of the spinal cord, with two main non-communicating feeding arteries, anterior and posterior. The anterior spinal artery is ultimately responsible for the risk of paraplegia, as it irrigates the anterior horns of the spinal cord. This artery is nourished, in a highly variable way, by a complex of aortic intercostal collateral vessels. The greatest of them is the great radicular artery (or artery of Adamkiewicz) originating somewhere between T6 and L1.

One of the most interesting aspects is why some people tolerate long periods of ischemia. This indicates that the cause is multifactorial, and it is based on three determinants: duration and degree of ischemia during aortic clamping, loss of nutritive flow in the anterior spinal artery (intercostal arteries), or postoperative events (reperfusion injury, hypotension).

SURGICAL TREATMENT (ST) OF AORTIC DISSECTION

The diagnosis of aortic dissection must determine whether the injury started in the ascending or descending aorta, since the prognosis varies considerably. The dissection that begins in the ascending aorta seriously compromises the patient's life because of the risk of pericardial rupture and cardiac tamponade. For this reason, this type of dissection is considered a surgical emergency. It is also imperative to eliminate aortic regurgitation and prevent myocardial ischemia.

Surgery should include 3 basic principles: resection of the intimal tear, the closing of the blood entry which produces the false lumen, and replacement of the damaged section of the aorta with a synthetic prosthesis.

It is also necessary to restore the competence of the aortic valve, which may be achieved with the resuspension technique.

Different variants of aortic dissection surgery include:

a) Patients with Type A aortic dissection, primary entry point in ascending aorta and aortic regurgitation, without affecting the sinuses of Valsalva and without poor peripheral perfusion: replacement of the ascending-hemiarch aorta and resuspension of the aortic valve, with hypothermia at 22 to 24 °C , circulatory arrest and antegrade cerebral perfusion via the right axillary artery. Approach through median sternotomy; the cannulation of the axillary artery has significantly reduced the incidence of neurological complications.
b) Patients with Type A aortic dissection, primary entry point in ascending aorta, impaired sinuses of Valsalva and aortic insufficiency, without poor peripheral perfusion: valved tube graft with reimplantation of coronary arteries and hemiarch, in circulatory arrest and antegrade cerebral perfusion via right axillary artery. In the absence of involvement of the aortic leaflets, a David or Yacoub procedure may be performed, with preservation of the aortic valve, replacement of sinuses of Valsalva and coronary artery reimplantation.
c) Patients with type A aortic dissection, primary entry point in descending aorta, without communication of both lumens in ascending aorta, no aortic valve involvement, no sinuses of Valsalva involvement and without poor peripheral perfusion: Replacement of the proximal descending thoracic aorta using deep hypothermia and circulatory arrest. Cannulation of femoral artery or left axillary artery, and femoro-atrial vacuum-assisted venous drainage. Left postero-lateral thoracotomy approach, through the fourth intercostal space.
   All patients with type A dissection, in any of its variants, with preoperative clinical or radiological evidence of poor peripheral perfusion, will undergo a diagnostic aortography after surgery; and, if needed, therapeutic aortography will be performed with stenting or fenestration.
   Yan *et al*[42] recently published the combined treatment of total replacement of the ascending aorta and the aortic arch, and the endovascular treatment of the descending aorta, in patients with type A dissection.
d) Patients with type B dissection complicated by persistent pain or increased left pleural effusion, without retrograde extension to the aortic arch: Replacement of the descending thoracic aorta

through left bypass (left atrium – aorta, or left atrium – femoral artery) or deep hypothermia and circulatory arrest, according to the possibilities for proximal or distal aortic clamping, or both. In the latter case, arterial cannulation in the aortic arch and femoro-atrial vacuum-assisted venous drainage must be performed. Distal anastomosis should be planned for preservation of the intercostal arteries T8-L2. The approach should be a left postero-lateral thoracotomy through the fourth intercostal space or double left postero-lateral thoracotomy through the fourth and seventh intercostal spaces.

e) Patients with type B dissection complicated by poor peripheral perfusion syndrome (clinical diagnosis or CT scan) with a very short time of progression: Emergency surgery using the technique of the previous example and postoperative diagnostic angiography, which could become a therapeutic procedure with stenting or fenestrations.
f) Patient with type B dissection complicated by poor peripheral perfusion syndrome, with hours of progression, or chronic type B aortic dissection with a new episode of redissection: diagnostic/therapeutic angiography with stenting and fenestration.

In young patients with type A dissection, it may be considered the replacement of the ascending aorta and the aortic arch through the elephant trunk technique, depending on the experience of the surgical team[41].

¿What are the factors of worse prognosis in the surgery of the ascending aorta?

These factors include being older than 70 years, hypotension, shock, cardiac tamponade, renal insufficiency or preoperative neurological damage, absence of peripheral pulse, ST abnormalities on electrocardiogram, previous cardiac surgery, preoperative myocardial infarction, visceral ischemia, concomitant lung disease, and prolonged extracorporeal circulation time[23,43,44].

Intramural hematoma and penetrating aortic ulcer will have the status of aortic dissection type A or B taking into account its location.

ENDOVASCULAR TREATMENT OF TYPE B DISSECTION

Dake *et al*[45], from Stanford University, published in 1999 the first case of the use of a stent to close the entry opening in a type B aortic dissection.

The 30-day mortality rate in patients with MT is 10 % and 19 % for those with ST[46]. However long term MT survival is 60-80 % at 4 or 5 years and 40-45 % at 10 years[21,47].

The relationship between survival and the patency of the false lumen was assessed in 201 patients included in the International Registry of Acute Aortic Dissection (IRAD) group[48]. Those who had a patent false lumen had better survival at 3 years than those with partially thrombosed lumen (14 vs. 32%).

The conventional ST, which shows a high mortality rate, should be used in the youngest patients, in good general condition, or in Marfan syndrome.

Uses

In recent years, endovascular surgery in type B dissection has simplified the approach and has decreased morbidity and mortalidad[49-52].

It would be used in the following situations:

- Prevention or treatment of rupture
- To avoid or block the progress of proximal or distal extension
- When there is only one entry opening
- To reduce the incidence of chronic aneurysm.

Although endovascular treatment is being used more frequently, the long-term results are still controversial[53-59]. The approach is different in the case of an acute, subacute or chronic dissection.

There is consensus that the treatment should be performed in patients with:

a) Refractory intractable pain despite aggressive antihypertensive treatment.
b) Signs of impending rupture detected by angiography or CT scan.
c) Significant false lumen with risk of forming a long-term aneurysm.
d) Patients with aortic diameters larger than 40 mm, which have worse long-term outcome.
e) Great periaortic or mediastinal hematoma.

Endovascular treatment should also be used in cases of poor perfusion of vital organs (renal or mesenteric), combining fenestrations or aorto-arterial bypass.

Before making any surgical decision, it is important to identify the anatomy of the dissection very well,

and make an appropriate medical and surgical plan. It is necessary to know the point of entry, possible re-entries, the extension of the dissected aorta, whether there is poor perfusion or not, and the state of the flow in the false lumen.

The CT scan, transesophageal echo and magnetic resonance imaging are useful; intravascular echo is also used, although it is limited to a few hospitals.

There is no consensus with regard to the appropriate moment to perform endovascular treatment; whether performing it in the acute phase or wait a few weeks to prevent future complications. It seems clear that early intervention is used in patients who have serious potential complications. If there are no complications, it is possible to wait 2 weeks.

A recent meta-analysis that includes 37 studies[53] analyzing the results of endovascular treatment with stents in 184 patients, using the IRAD data base, shows better results for endovascular treatment compared to MT and ST. In 2006, the results of 242 patients with aortic dissection type B were published[53]; 78% of the cases received MT, 11% ST and 11% endovascular treatment, with an overall mortality of 12%. Survival at 3 years was similar in the 3 groups.

FOLLOW UP

Survival of patients who underwent surgery is 52 % at 10 years[14].

It is important to follow up closely the patients and control the risk factors, particularly BP, since about 30 % of patients have progression of the size of the aneurysm or new dissections.

The first CT scan should be performed 3-4 months after hospital discharge to rule out the possibility that the patient has a rapid growth of the aorta. If the size is 35-40 mm, the monitoring should be every 12 months, if the size is 45-50 mm, monitoring should be every 6 months. In patients with impaired renal function, the non-contrast CT scan may be sufficient.

The optimal treatment of aortic dissection is highly variable due to multiple factors. In those patients receiving medical management and in those undergoing surgical treatment, all kinds of complications may be expected.

In cases of chronic aortic dissection, some authors[54] measure the growth rate of the diameter of the aorta by magnetic resonance imaging performed every 12 months in stable patients. If the diameter exceeds 5-7 mm during this period of time, it is interpreted as a state of instability and it is treated with stents. The consensus for the treatment of chronic aortic dissection is similar to other causes of aortic aneurysms (atherosclerotic, mycotic or traumatic causes). Aneurysms over 5.5 cm on the ascending aorta and over 6 cm on the descending aorta are in danger of rupture and it is necessary to perform surgery or stenting.

At one year, 95% of patients remain without intervention, 90 % at 5 years, 74 % at 10 years and 65 % at 15 years[47,59,60].

Risk factors that predict the need for reoperation

a) Marfan Syndrome
b) The entry opening has not been occluded
c) Lack of beta blockers in the postoperative period
d) Uncontrolled hypertension.

Other authors[43,44,47,48] recommend reoperation for chronic aortic dissection on the descending aorta when:

e) The aortic diameter is over 6.0 to 6.5 cm.
f) There is a growth greater than 5-7 mm in a year.
g) There is recurrent pain despite tight control of blood pressure.
h) There is saccular dilatation with danger of rupture.
i) In patients with Marfan syndrome with a growth greater than 3-5 mm/year.

RECOMMENDATIONS

The European Society of Cardiology published in the year 2001[61] the following recommendations:

- Initial treatment in type A aortic dissection: beta blockers, and analgesics. Perform transthoracic or transesophageal echocardiography and call the surgeon. The CT scan is the most commonly used technique in the diagnosis of aortic dissection, with a sensitivity over 90% and a specificity of 85 %.
- MT is administered if the echocardiogram shows tamponade. If there is aortic insufficiency, valve resuspension. In case of aneurysm of the sinuses of Valsalva together with aortic dissection (Marfan), valved tube placement. If there is an obstruction of one of the branches of the aorta, stent placement or fenestration.
- Treatment of type B aortic dissection: MT is the treatment of choice, if there are complications, such as early expansion, recurrent pain, ischemic

involvement or rupture, surgical treatment will be used.
- In chronic dissection, the use of magnetic resonance imaging is advisable. Angiography is indicated in patients who will undergo percutaneous intervention.
- If prosthesis infection appears, a resection of all infected tissue is required, and the placement of a homograft.

Later, in 2010, the guidelines for the diagnosis and management of patients with thoracic aortic disease, approved by the ACCF/AHA/AATS/ACR/ASA/SCAI/SIR/STS/SVM[62], were published:

Patients suspected of having aortic dissection: risk patients are those with a history of Marfan syndrome, Ehlers-Danlos syndrome, Loeys-Dietz syndrome, etc; genetic mutations in FB1, TGFBR1, ACTA2 and MYH11 genes; a family history of aortic aneurysm, recent handling of the aorta. Patients with anterior and posterior chest pain, or intense sudden abdominal pain, associated with pulse deficit or neurological disorders with recent-onset aortic regurgitation murmur.

The guidelines[62] cover extensively the diagnosis and treatment of all aneurysms, including dissections.

CONCLUSIONS

Acute aortic dissection is a disease with high mortality and morbidity rates if it is not treated properly and early. Aortic dissection should be suspected in patients with severe chest pain, hypertension and normal or inconclusive electrocardiogram. Surgical treatment should not be delayed in patients with a diagnosis of type A aortic dissection, since mortality depends on time. In type B aortic dissection, medical treatment with strict control of blood pressure and beta-blockers is advisable, reserving surgical management, either endovascular or surgical treatment, for patients with complications.

REFERENCES

1. Nienaber CA, Eagle KA. Aortic dissection: New frontiers in diagnosis and management. Part I: From etiology to diagnostic strategies. Circulation. 2003;108(5):628-35.
2. Mészáros I, Mórocz J, Szlávi J, Schmidt J, Tornóci L, Nagy L, et al. Epidemiology and clinicopathology of aortic dissection. Chest. 2000;117(5):1271-8.
3. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA. 2000;283(7): 897-903.
4. Fann JI, Sarris GE, Mitchell RS, Shumway NE, Stinson EB, Oyer PE, et al. Treatment of patients with aortic dissection presenting with peripheral vascular complications. Ann Surg. 1990;212(6):705-13.
5. Moon MR, Sundt TM, Pasque MK, Barner HB, Huddleston CB, Damiano RJ, et al. Does the extent of proximal or distal resection influence outcome for type A dissections? Ann Thorac Surg. 2001; 71(4):1244-9.
6. Lauterbach SR, Cambria RP, Brewster DC, Gertler JP, Lamuraglia GM, Isselbacher EM, et al. Contemporary management of aortic branch compromise resulting from acute aortic dissection. J Vasc Surg. 2001;33(6):1185-92.
7. Zierer A, Moon MR, Melby SJ, Moazami N, Lawton JS, Kouchoukos NT, et al. Impact of perfusion strategy on neurologic recovery in acute type A aortic dissection. Ann Thorac Surg. 2007;83(6): 2122-8.
8. DeBakey ME, Crawford ES, Garrett HE, Beall AC, Howell JF. Surgical consideration in the treatment of aneurysms of the thoraco-abdominal aorta. Ann Surg. 1965;162(4):650-62.
9. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. Ann Thorac Surg. 1970;10(3):237-47.
10. Sheikh AS, Ali K, Mazhar S. Acute aortic syndrome. Circulation. 2013;128(10):1122-7.
11. von Kodolitsch Y, Csösz SK, Koschyk DH, Schalwat I, Loose R, Karck M, et al. Intramural hematoma of the aorta: predictors of progression to dissection and rupture. Circulation 2003;107(8):1158-63.
12. David P, McPeak EM, Vivas-Salas E, White PD. Dissecting aneurysm of the aorta: A review of 17 autopsied cases of acute dissecting aneurysm of the aorta encountered at the Massachusetts General Hospital from 1937 to 1946 inclusive, eight of which were correctly diagnosed ante mortem. Ann Intern Med. 1947;27(3):405-19.
13. Borst HG, Heinemann MK, Stone CH. Surgical treatment of aortic dissection. New York: Churchill Livingstone, 1996.
14. Olsson Ch, Thelin S, Ståhle E, Ekbom A, Granath F.

Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14.000 cases from 1987 to 2002. Circulation. 2006; 114(24):2611-8.
15. Anagnostopoulos CE. Acute aortic dissection. Baltimore: University Park Press, 1975.
16. Appelbaum A, Karp RB, Kirklin JW. Ascending vs descending aortic dissections. Ann Surg. 1976; 183(3):296-300.
17. Borger MA, Preston M, Ivanov J, Fedak PW, Davierwala P, Armstrong S, et al. Should the ascending aorta be replaced more frequently in patients with bicuspid aortic valve disease? J Thorac Cardiovasc Surg. 2004;128(5):677-83.
18. Gott VL, Pyeritz RE, Magovern GJ, Cameron DE, McKusick VA. Surgical treatment of aneurysms of the ascending aorta in the Marfan syndrome. Results of composite-graft repair in 50 patients. N Engl J Med. 1986;314(17):1070-4.
19. Isselbeuber EM, Eagle KA, Desanctis RW. Diseases of the aorta. En: Braunwald E. Heart diseases. Philadelphia: Saunders, 1997; p. 1546-81.
20. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, et al. Diagnosis and management of aortic dissection. Eur Heart J. 2001;22(18):1642-81.
21. Doroghazi RM, Slater EE, DeSanctis RW, Buckley MJ, Austen WG, Rosenthal S. Long-term survival of patients with treated aortic dissection. J Am Coll Cardiol. 1984;3(4):1026-34.
22. Glower DD, Fann JI, Speier RH, Morrison L, White WD, Smith LR, et al. Comparison of medical and surgical therapy for uncomplicated descending aortic dissection. Circulation 1990;82(5 Suppl):IV39-46.
23. Miller DC. Acute dissection of the descending aorta: clinical dilemma and hospital perspective. Chest Surg Clin North Am. 1992;2:347-55.
24. Miller DC. The continuing dilemma concerning medical versus surgical management of patients with acute type B dissections. Semin Thorac Cardiovasc Surg. 1993;5(1):33-46.
25. Vedantham S, Picus D, Sanchez LA, Braverman A, Moon MR, Sundt T, et al. Percutaneous management of ischemic complications in patients with type-B aortic dissection. J Vasc Interv Radiol. 2003; 14(2 Pt 1):181-94.
26. Jonker FH, Trimarchi S, Muhs BE, Rampoldi V, Montgomery DG, Froehlich JB, et al. The role of age in complicated acute type B aortic dissection. Ann Thorac Surg. 2013;96(6):2129-34.
27. Shores J, Berger KR, Murphy EA, Pyeritz RE. Progression of aortic dilatation and the benefit of long-term beta-adrenergic blockade in Marfan's syndrome. N Engl J Med. 1994;330(19):1335-41.
28. Kazui T, Washiyama N, Muhammad BA, Terada H, Yamashita K, Takinami M. Improved results of atherosclerotic arch aneurysm operations with refined technique. J Thorac Cardiovasc Surg. 2001;121(3): 491-9.
29. Bonow RO, Cheitlin MD, Crawford MH, Douglas PS. Task force 3: valvular heart disease. J Am Coll Cardiol. 2004;45(8):1334-40.
30. Maron BJ, Ackerman MJ, Nishimura RA, Pyeritz RE, Towbin JA, Udelson JE. Task Force 4: HCM and other cardiomyopathies, mitral valve prolapse, myocarditis, and Marfan syndrome. J Am Coll Cardiol. 2005;45(8):1340-5.
31. Comas GM, Leshnower BG, Halkos ME, Thourani VH, Puskas JD, Guyton RA, et al. Acute type A dissection: impact of antegrade cerebral perfusion under moderate hypothermia. Ann Thorac Surg. 2013;96(6):2135-41.
32. Baribeau YR, Westbrook BM, Charlesworth DC, Maloney CT. Arterial inflow via an axillary artery graft for the severely atheromatous aorta. Ann Thorac Surg. 1998;66(1):33-37.
33. Whitlark JD, Goldman SM, Sutter FP. Axillary artery cannulation in acute ascending aortic dissections. Ann Thorac Surg. 2000;69(4):1127-9.
34. Etz CD, von Aspern K, da Rocha J, Girrbach FF, Leontyev S, Luehr M, et al. Impact of perfusion strategy on outcome after repair for acute type A aortic dissection. Ann Thorac Surg. 2014;97(1):78-85.
35. Ueda Y, Miki S, Kusuhara K, Okita Y, Tahata T, Yamanaka K. Deep hypothermic systemic circulatory arrest and continuous retrograde cerebral perfusion for surgery of aortic arch aneurysm. Eur J Cardiothorac Surg. 1992;6(1):36-42.
36. Higami T, Kozawa S, Asada T, Obo H, Gan K, Iwahashi K, et al. Retrograde cerebral perfusion versus selective cerebral perfusion as evaluated by cerebral oxygen saturation during aortic arch reconstruction. Ann Thorac Surg. 1999;67(4):1091-6.
37. Hilgenberg AD. Spinal cord protection for thoracic aortic surgery. Cardiol Clin. 1999;17(4):807-13.
38. Kouchoukos NT, Rokkas CK. Hypothermic cardiopulmonary bypass for spinal cord protection: ratio-

nale and clinical results. Ann Thorac Surg. 1999; 67(6):1940-2.
39. Safi HJ, Miller CC. Spinal cord protection in descending thoracic and thoracoabdominal aortic repair. Ann Thorac Surg. 1999;67(6):1937-9.
40. Svensson LG. An approach to spinal cord protection during descending or thoracoabdominal aortic repairs. Ann Thorac Surg. 1999;67(6):1935-8.
41. Bachet J1, Guilmet D, Goudot B, Dreyfus GD, Delentdecker P, Brodaty D, et al. Antegrade cerebral perfusion with cool blood: a 13-year experience. Ann Thorac Surg. 1999;67(6):1891-4.
42. Yan Z, Shen YH, Zhang QC, Zhu ZY, Lei H, Cheng GC, et al. A combined branched stent grafting technique to reconstruct total aortic arch in the treatment of Stanford A aortic dissection. Ann Thorac Surg. 2013;96(5):1888-90.
43. Tsai TT, Evangelista A, Nienaber CA, Myrmel T, Meinhardt G, Cooper JV, et al. Partial thrombosis of the false lumen in patients with acute type B aortic dissection. N Engl J Med. 2007;357(4):349-59.
44. Mehta RH, O'Gara PT, Bossone E, Nienaber CA, Myrmel T, Cooper JV, et al. Acute type A aortic dissection in the elderly: clinical characteristics, management and outcomes in the current era. J Am Coll Cardiol. 2002;40(4):685-92.
45. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, Shimono T, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med. 1999;340(20):1546-52.
46. Glower DD, Fann JI, Speier RH, Morrison L, White WD, Smith LR, et al. Comparison of medical and surgical therapy for uncomplicated descending aortic dissection. Circulation. 1990;82(5 Suppl):IV39-46.
47. Borst HG, Jurmann MJ, Heinemann MK, Laas J: Redo surgery after primary operation for acute and chronic aortic dissection. International symposium on diseases of the aorta. Tokio, 1992; p. 28. [Resumen].
48. Umaña JP, Lai DT, Mitchell RS, Moore KA, Rodriguez F, Robbins RC, et al. Is medical therapy still the optimal treatment strategy for patients with acute type B aortic dissections? J Thorac Cardiovas Surg. 2002;124(5):896-910.
49. Tan WA. Endovascular treatment of aortic dissections. Catheter Cardiovasc Interv. 2003;58(1):101-2.
50. Mitchell RS, Ishimaru S, Ehrlich MP, Iwase T, Lauterjung L, Shimono T, et al. First International Summit on Thoracic Aortic Endografting: roundtable on thoracic aortic dissection as an indication for endografting. J Endovasc Ther. 2002;9(Suppl 2):II98-105.
51. Nienaber CA, Ince H, Petzsch M, Rehders T, Körber T, Schneider H, et al. Endovascular treatment of thoracic aortic dissection and its variants. Acta Chir Belg. 2002;102(5):292-8.
52. Schütz W, Gauss A, Meierhenrich R, Pamler R, Görich J. Transesophageal echocardiographic guidance of thoracic aortic stent-graft implantation. J Endovasc Ther 2002;9(Suppl 2):II14-9.
53. Eggebrecht H, Nienaber CA, Neuhäuser M, Baumgart D, Kische S, Schmermund A, et al. Endovascular stent-graft placement in aortic dissection: a meta analysis. Eur Heart J. 2006;27(4):489-98.
54. Tsai TT, Fattori R, Trimarchi S, Isselbacher E, Myrmel T, Evangelista A, et al. Long-term survival in patients presenting with type B acute aortic dissection: insights from the International Registry of Acute Aortic Dissection. Circulation. 2006;114(21): 2226-31.
55. White RA, Donayre C, Walot I, Lee J, Kopchok GE. Regression of a descending thoracoabdominal aortic dissection following staged deployment of thoracic and abdominal aortic endografts. J Endovasc Ther. 2002;9(Suppl 2):II92-7.
56. Criado FJ, Barnatan MF, Rizk Y, Clark NS, Wang CF. Technical strategies to expand stent-graft applicability in the aortic arch and proximal descending thoracic aorta. J Endovasc Ther. 2002;9(Suppl 2): II32-8.
57. Görich J, Asquan Y, Seifarth H, Krämer S, Kapfer X, Orend KH, et al. Initial experience with intentional stent-graft coverage of the subclavian artery during endovascular thoracic aortic repairs. J Endovasc Ther. 2002;9(Suppl 2):II39-43.
58. Shim WH, Koo BK, Yoon YS, Choi D, Jang Y, Lee DY, et al. Treatment of thoracic aortic dissection with stent-grafts: midterm results. J Endovasc Ther. 2002;9(6):817-21.
59. Pamler RS, Kotsis T, Görich J, Kapfer X, Orend KH, Sunder-Plassmann L. Complications after endovascular repair of type B aortic dissection. J Endovasc Ther. 2002;9(6):822-8.
60. Chiappini B, Schepens M, Tan E, Dell' Amore A, Morshuis W, Dossche K, et al. 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.
61. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, et al. Diagnosis and management of aortic dissection. Eur Heart J. 2001;22(18):1642-81.
62. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the diagnosis and management of patients with thoracic aortic disease. A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology,American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons,and Society for Vascular Medicine. J Am Coll Cardiol. 2010;55(14):e27-e129.