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Aortic interlude: Dr Michael DeBakey, aortic dissection, and screening recommendations for abdominal aortic aneurysm

A visionary surgeon's development of functional Dacron grafts in the 1950s has made effective treatment of aortic dissection and aneurysm possible.

ABSTRACT: Acute aortic dissection remains one of the most common fatal conditions not diagnosed correctly by physicians. It results from a tear in the aorta separating the intimal layer from the medial layer, and is associated with age, hypertension, collagen vascular disease, and other factors. It frequently presents with atypical symptoms, and physical examination is often unhelpful. Diagnosis requires contrast CT, MRI, or transesophageal echocardiography. Dissections that involve the ascending aorta require emergency surgery, whereas those involving the descending aorta can be managed initially with close observation and blood pressure control. Physicians can learn much from the life and accomplishments of Michael DeBakey, one of the early pioneers in managing aortic dissection. They can also benefit from reviewing the European Society of Cardiology 2001 guidelines for the diagnosis and management of aortic dissection, and the American Heart Association 2005 guidelines for aortic aneurysm screening.

This article has been peer reviewed.

Dr Michael DeBakey

Michael Ellis Dabaghi was born in Lake Charles, Louisiana, on 7 September 1908. He was the eldest of four children of Lebanese immigrants, and his surname was later anglicized to "DeBakey." His father was the proprietor of two successful drug stores, where DeBakey's interest in medicine may have been ignited by the physicians who patronized the establishments. Prior to enrolling in high school, DeBakey demonstrated both tenacity and diligence by reading the entire *Encyclopedia Britannica*.

At the age of 20 he enrolled in medical school at Tulane University in New Orleans. At Tulane he met the legendary surgeon Alton Ochsner, who recognized the eager medical student's potential and fostered his interest in surgery. Together they were among the first to propose a link between smoking and lung cancer. Still as a medical student, DeBakey developed one of the first mechanical blood pumps—the "roller pump"—which enabled the transfer of blood along a conduit. This same pump would become an integral component of the heart-lung machine that ushered in the era of cardiac surgery.

After completing medical school in 6 years—a task that normally took 8 years—he trained as a surgeon at the universities of Strasbourg and Heidelberg. In 1938 he returned to Tulane to accept a faculty position in general surgery. During the Second World War he served in the Surgeon General's Office, where he rose to the rank of colonel and become the chief of the surgical consultants division. After the war, DeBakey participated in the Hoover Commission—an organization created by President Truman to recommend administrative changes to the federal government of the United States. His lobbying for a national library of medical articles ultimately led to the creation of the National Library of Medicine in 1959. In later years DeBakey would serve as its chairman.

In 1948 DeBakey moved to Houston, Texas, where he took a position as professor of surgery at the then mediocre Baylor College of Medicine. There DeBakey was in the forefront of the nascent field of vascular

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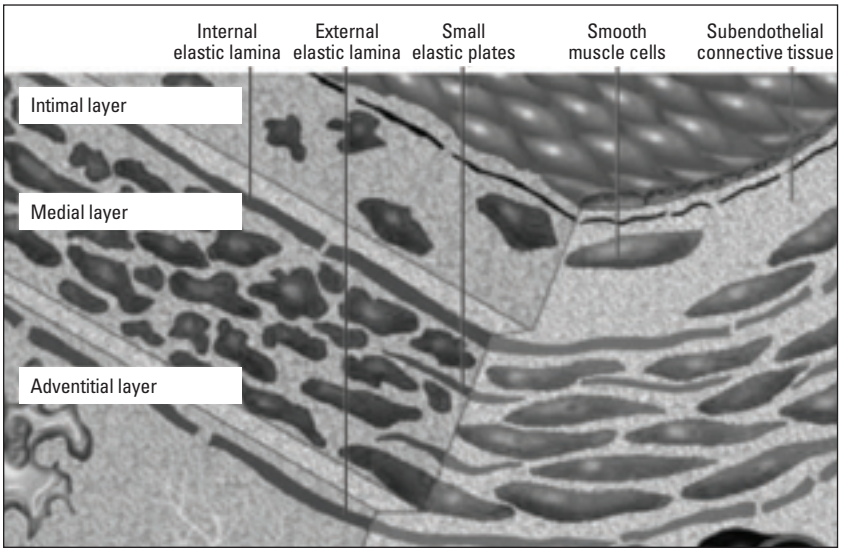

Figure 1. Schematic of arterial wall, showing layers ranging from deep (intima) to superficial (adventitia).

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surgery. In 1952 he was the first to repair an aortic aneurysm using a cadaver graft. The next year he was the first to successfully perform a carotid endarterectomy. He then began to experiment with the creation of grafts for aortic surgery. DeBakey's colleagues had attempted to build nylon grafts, but found that they deteriorated in vivo. On one occasion, when purchasing new material for his experiments, he found the local department store out of nylon, but in receipt of a new fabric known as Dacron. Using his mother's techniques and his wife's sewing machine, he was able to turn this new material into arterial patches—the first functional Dacron grafts. These grafts would go on to revolutionize the treatment of aortic pathology. Over the next several years Dr DeBakey was instrumental in the development of surgical techniques for the repair of aortic dissection and aortic aneurysms.

In the 1960s DeBakey and his team began to focus on the development of artificial hearts and cardiopulmonary assist devices. In 1963 DeBakey created the first pneumatically powered ventricular assist device (VAD). It was implanted in a cardiogenic shock patient and provided support for 4 days before the patient died from causes unrelated to the device. His team's VAD designs were refined over the next decade. This culminated in smaller, continuous-flow devices that could be implanted in a patient, to permit chronic mechanical cardiac support.

DeBakey's many other accomplishments are too numerous to list. They include the American Medical Association's Distinguished Service Award in 1959, the Albert Lasker Clinical Medical Research Award in 1963, the Presidential Medal of Freedom in 1969, and the National Medal of Science in 1987. In 2000 he was recognized as a Living Legend by the US Library of Congress. In 2008 he received the Congressional Gold Medal, making him only the third physician to have received that honor.

DeBakey continued to be a driving force in surgical education and administration well into his 90s, serving as Chancellor Emeritus of Baylor College of Medicine. In 2006 he had the unique experience of benefiting from a procedure that he had devised. At the age of 97 he developed a sharp tearing chest pain as he was preparing for a lecture. He was rushed to Methodist Hospital in Houston, where a CT scan confirmed the diagnosis of a type A aortic dissection. Because his advanced age made surgery perilous, his condition was initially managed medically, and against the advice of his physicians he went on to give his lecture only a week after the diagnosis was made. Unfortunately, the dissection continued to progress and DeBakey was readmitted to hospital within a month. After some discussions regarding the ethics of proceeding, the decision was made to operate. The repair was performed by Dr George Noon, DeBakey's long-time assistant. The tenacious DeBakey recovered well despite his age, and went on to live another two years. He died suddenly from natural causes at the age of 99 on 11 July 2008, having revolutionized the management of aortic dissection.

Pathophysiology and epidemiology

In North America the incidence of aortic dissection is estimated to be approximately 2.9 cases per 100 000 patient years.[1] Dissection occurs most commonly in patients aged 60 to 80, and is slightly more common in men.[2,3] It is also associated with hypertension and collagen vascular disease.

The aortic wall consists of three layers: the intima, the media, and the adventitia (**Figure 1**).[4] Classic aortic dissection occurs when a tear in the intima permits the entry of blood to a diseased underlying media, causing the separation of the intima and media

Table 1. Risk factors identified by the International Registry of Acute Aortic Dissection (IRAD).[3]

	Age > 70	Male sex	Hyper-tension	Athero-sclerosis	Previous cardiac surgery	Aortic aneurysm	Marfan syndrome	Iatro-genic	Bicuspid aortic valve	Diabetes	Previous dissection	Preg-nancy	Cocaine use
Type A dissection (n = 617)	194 (31%)	413 (67%)	408 (67%)	169 (28%)	100 (16%)	42 (7%)	38 (6%)	34 (6%)	14 (4%)	24 (4%)	21 (3%)	1 (<1%)	1 (< 1%)
Type B dissection (n = 384)	159 (42%)	274 (71%)	303 (80%)	140 (38%)	62 (17%)	68 (18%)	11 (3%)	9 (2%)	4 (2%)	24 (7%)	33 (9%)	1 (< 1%)	4 (1%)
Dissection in patients < 50 years (n = 68)	n/a	52 (76%)	23 (34%)	1 (1%)	8 (12%)	13 (19%)	34 (50%)	0	6 (9%)	0	5 (7%)	2 (3%)	0

and the formation of a false lumen.[5] This false lumen can extend proximally or distally, and may compromise branch vessels and lead to end organ ischemia. Alternatively, the dissection may extend into the pericardium leading to cardiac tamponade.

In the 1950s Dr Michael DeBakey made a major contribution to the management of aortic pathology by pioneering the use of arterial patches. The grafting techniques these made possible allowed for improved repair of aortic dissections and aneurysms.

Risk factors for aortic dissection

The International Registry of Acute Aortic Dissection (IRAD) is the authority on risk factors for this arterial pathology. The most common acquired condition predisposing patients to dissection is chronic hypertension, which is present in 75% of cases.[2] Elevated arterial pressures lead to intimal thickening, fibrosis, and calcification. This limits blood supply to the arterial wall. In addition, the extracellular matrix is modified with enhanced apoptosis and elastolysis.[5] Atherosclerosis leads to adventitial fibrosis and compromise of the vasa vasorum—the small vessels that penetrate into the vessel wall. All of the above lead to necrosis of the smooth muscle cells and fibrosis of elastic structures in the media, resulting in stiffness, weakness, and a vulnerability to shear forces. With time, this leads to aneurismal dilation and dissection.

Patients with inherited defects of connective tissue synthesis, such as Marfan syndrome, Ehlers-Danlos syndrome, and annuloaortic ectasia, are predisposed to degeneration and elastolysis of the aortic media.[5] As a consequence they are at risk for aortic aneurysm and dissection at a young age. In the IRAD registry, patients with inherited connective tissue disorders account for more than 50% of dissections occuring at less than 40 years of age.[2] Other acquired risk factors include atherosclerosis, inflammatory conditions causing aortitis (e.g., Takayasu arteritis, giant cell arteritis, syphilis), previous cardiac surgery, bicuspid aortic valve, and cocaine use. Some of the risk factors for dissection found in the IRAD registry are shown in **Table 1**.

Staging

Several different staging systems have been used to stratify aortic dissection. In the Stanford system, type A dissections involve the ascending aorta, while type B dissections involve only the descending aorta (**Figure 2**).[3,6,7] In

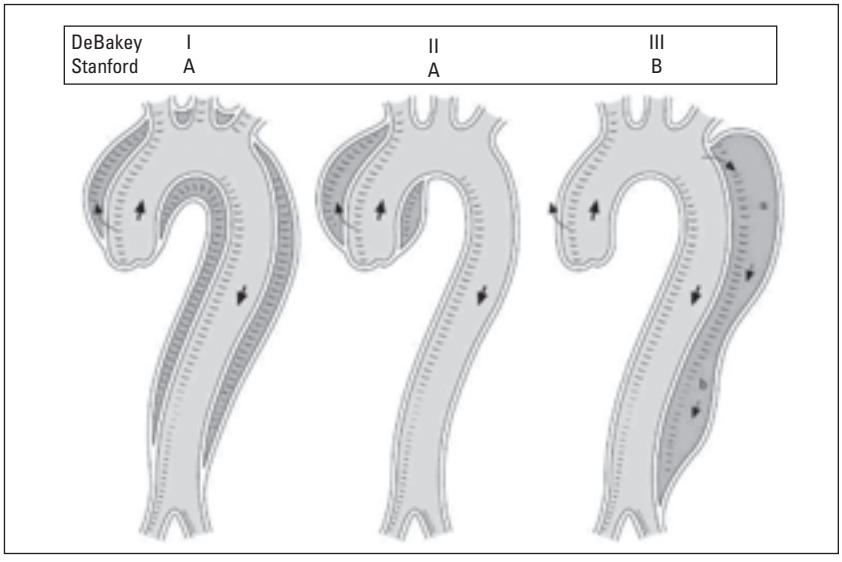

Figure 2. The Stanford and DeBakey classification systems for aortic dissection.

From Erbel R, Alfonso F, Boileau C, et al.[7] Reproduced with permission.

Table 2. ESC recommendations for imaging modalities in suspected aortic dissection.

Modality	Advantages	Disadvantages	Level of evidence
TTE/TEE	• Fast, accurate • Can be done at bedside • Can quantify AI, EF	• Spatial resolution low • Blind spot	Class I
CT angiography	• Fast, accurate	• Cannot quantify AI, EF • Radiation, contrast exposure	Class I
MR angiography*	• Best resolution • Can quantify AI, EF	• Requires breath holding • Limited availability	Class IIa
Contrast angiography*	• Useful to guide percutaneous intervention	• Low sensitivity • Resource intensive • Radiation, contrast exposure	Class IIa

AI = aortic insufficiency; EF = ejection fraction; TEE = transthoracic echocardiography
TTE = transesophageal echocardiography; *Not recommended for unstable patients

the DeBakey classification system, a type I dissection involves the entire aorta, while a type II dissection involves only the ascending aorta, and a type III dissection involves only the descending aorta. New staging systems that incorporate other aortic pathology have been devised, but are used primarily for research purposes.

Clinical presentation

The primary presenting complaint in aortic dissection is chest pain. The pain is typically described as severe and abrupt in onset (90%), and the character is sharp as opposed to tearing, ripping, or stabbing.[8] The absence of pain does not rule out dissection, as 10% of cases are asymptomatic on presentation.[3] In type A dissections, 25% of patients will present in shock, in contrast to type B dissections where hypertension is a common finding.[3] Up to 20% of patients may present with syncope or with neurological symptoms.[5] Abdominal pain suggests involvement of the celiac artery with mesenteric ischemia, anuria suggests involvement of the renal arteries, and lateralizing neurological signs suggest compromise of the carotids.[7]

Physical examination often contributes little to findings. Although pulse deficits are suggestive of dissection, these are present in only 15% of type A dissections.[9] Neurological signs are present in 40% of proximal dissections.[7] The early diastolic murmur of acute aortic insufficiency may be present in 30% to 50% of type A dissection cases; however, the murmur is typically soft and peripheral signs of aortic insufficiency are absent. Pericardial friction rub, elevated jugular venous pressure, or pulsus paradoxus are infrequent but suggest a worrisome extension of the dissection into the pericardium—a condition that can be rapidly fatal. Because the clinical presentation is nonspecific, the diagnosis is frequently missed. In one review from the Mayo Clinic, the correct diagnosis was not made prior to postmortem examination in 28% of cases.

Investigations

In 2001 a taskforce of the European Society of Cardiology (ESC) published guidelines regarding the diagnosis and management of aortic dissection.[7] These guidelines remain the most recent of any produced by an international society.

Acceptable imaging modalities include CT angiography, transthoracic echocardiography (TTE) followed by transesophageal echocardiography (TEE), MRI, and contrast angiography. The choice of imaging modality depends greatly on availability, and on the experience of the emergency room and emergency staff with the modality. The ESC 2001 recommendations for diagnostic imaging in suspected aortic dissection are summarized in **Table 2**.

The availability of computed tomography and its high sensitivity and specificity makes it the most commonly used modality in suspected aortic dissection.[7] CT provides accurate assessment of branch vessel involvement.[10] Limitations include the fact that aortic insufficiency and left ventricular function cannot be quantified, and the modality is associated with radiation exposure and potentially nephrotoxic contrast.

Echocardiography is useful in that it can be performed portably at the bedside or in the operating room if the patient is unstable. Transesophageal echocardiography needs to be performed in addition to transthoracic echocardiography in order to identify distal dissections.[7] Echocardiography allows quantification of aortic regurgitation and left ventricular function. The major disadvantage is poor spatial resolution and blind spots caused by intervening anatomical structures.[11]

Cardiac MR imaging provides very detailed images of the proximal and ascending aorta and also permits the quantification of aortic insufficiency and left ventricular function.[7] Radiation and contrast exposure is also limited with this modality. The disadvantage is the limited availability of MRI and the need for the patient to tolerate breath holding.[7,11]

Prior to the development of the above imaging modalities, contrast aortography was the criterion standard for diagnosing dissection. It is now performed infrequently because of its low sensitivity, its invasiveness, and the time delays associated with

activating an angiography suite.[7] It is useful, however, for identifying side branch occlusion and offers the possibility of percutaneous intervention to restore blood flow.

Management

The initial management of aortic dissection requires transfer to a critical care setting, pain control, and blood pressure control.[7] The patient should be placed on telemetry, and invasive blood pressure monitoring should be initiated (Class I evidence). Morphine sulphate is the drug of choice for pain and complete control should be the goal (Class I). Blood pressure should be kept in the range of 100 to 120 mm Hg through the use of intravenous medications.[7,12] Initial blood pressure control should be achieved through the use of beta blockers followed by vasodilators (Class I). Beta blockers decrease the shear stress on the wall of the aorta (dP/dt), thus they should be initiated before vasodilators. Labetalol is used frequently as it also has vasodilatory effects and thus is more effective at lowering the blood pressure. Pharmacological options for the management of acute dissection are listed in **Table 3**.[13]

Table 3. Pharmacological therapies for initial management of blood pressure in acute aortic dissection.

	Medication	Dosage	Onset of action	Duration
Beta blocker therapy	Labetalol	• 20–80 mg IV bolus every 10 minutes • 0.5-2.0 mg/min IV infusion	5–10 minutes	3–6 hours
	Esmolol	• 250–500 µg/kg/min by infusion • May repeat bolus after 5 minutes or increase infusion to 300 µg/min	1–2 minutes	10–30 minutes
Vasodilator therapy	Nitroprusside	• 0.25–10.00 µg/kg/min IV infusion	Immediate	1–2 minutes
	Hydralazine	• 10–20 mg IV bolus	10–20 minutes	1–4 hours
	Nitroglycerin	• 5–100 µg/min IV infusion	2–5 minutes	5–10 minutes

Type A dissections

Type A dissections carry the highest risk of aortic rupture, branch vessel occlusion, and cardiac tamponade, and emergency surgery is recommended in dissections of this type[7] (Class I). Various surgical procedures have been developed for the management of proximal aortic dissection, yet they all involve replacing the involved segment with a graft. If the aortic valve is structurally normal, and if there is no significant aortic insufficiency or dilation of the aortic root, then valve-preserving surgery is usually possible. This involves the placement of a tubular graft anastamosed to the sinotubular ridge.[7] If the aortic valve is incompetent or if the aortic root is ectatic, then replacement of the valve is required in addition to the placement of a graft. Recently, valve-sparing operations and root-remodeling procedures have been investigated, but these procedures are more difficult technically.[7] Currently, valve-sparing and root-remodeling procedures receive a Class IIa recommendation from the ESC.

Type B dissections

Uncomplicated Type B dissections are managed medically with blood pressure control and close observation. Surgical intervention is limited to the management of life-threatening complications or intractable symptoms. According to the ESC guidelines, surgery is recommended for unmanageable pain, aortic expansion, and periaortic or mediastinal hematoma that may herald aortic rupture.[7] The surgical management in type B dissection involves replacing the involved segment of the aorta with a graft.

Acute ischemia of the limbs, kidneys, or gut secondary to occlusion of an aortic branch may be managed initially by percutaneous catheter-based fenestration of the dissection flap, followed in some cases by stenting of the true lumen. If catheter-based therapies do not immediately restore perfusion to the ischemic organ, emergency surgery is indicated. In one case series, percutaneous intervention was able to restore blood flow to the compromised organ in 90% of cases.[7]

Outcomes

Today aortic dissection is no longer the almost uniformly fatal diagnosis it once was. In one recent study, surgically treated type A dissection was associated with a 22% in-hospital mortality rate but a long-term survival of 95% at 5 years and 88% at 10 years.[14] The IRAD registry found that in-hospital mortality was 13% for type B dissections, with most deaths occurring in the first week.[15] Patients managed medically alone had an in-hospital mortality rate of 10% compared with a 32% rate for those undergoing surgery. The higher mortality rate for surgical patients reflects the fact that their dissections were complicated and more extensive than those managed medically. Long-term survival rates of 60% at 5 years and 35% at 10 years have recently been reported for type B dissections.[16]

Long-term follow-up

When following up dissection patients it is important to realize that they have

Table 4. ESC guidelines for follow-up of patients with prior aortic dissection.

Intervention	Recommendations	Level of evidence
Medical therapy	Lifelong beta blockade	Class II
	Control of BP to less than 135/80 mm Hg (130/80 for Marfan syndrome)	Class I
Surveillance	Imaging at 1, 3, 6, and 12 months, then yearly therafter (by MRI, CT, or transthoracic echocardiography)	Class I
Repeat aortic surgery	Surgery for secondary aneurysm in dissected aorta remote from initial repair	Class I
	Surgery for recurrent dissection or aneurysm formation at previous intervention site	Class I
	Use of homografts to replace infected prostheses	Class IIa
	Endovascular stenting instead of surgery if anatomy is suitable	Class IIa

a systemic disease and are at risk for further events. It is estimated that one-third of dissection patients will experience dissection extension or aortic rupture, or will require repeat aortic surgery within 5 years of their index event.[7] In order to minimize wall stress, continued beta blocker therapy is required. Guidelines recommend a blood pressure of less than 130/80 mm Hg for patients with Marfan syndrome, and less than 135/80 mm Hg for all other patients (Class I). The avoidance of strenuous physical activity is also recommended, especially for patients with Marfan syndrome. Examples of activities to avoid include weight lifting, body building, and contact sports such as football and ice hockey.[17]

Patients with prior dissection require serial imaging. The goal of serial imaging is to detect extension or recurrence of the dissection, aneurysm formation, or leakage at anastamoses or stent sites. TEE and CT are acceptable, though MRI is the imaging modality of choice, as it is less invasive than TEE and does not expose the patient to radiation or nephrotoxic contrast agents. Repeat imaging of the aorta should be performed at 1, 3, 6, and 12 months following discharge and then on a yearly basis. Of course, in the setting of recurrent chest pain, repeat imaging should be obtained on an emergency basis.

Repeat aortic surgery is required when a dissected segment becomes aneurismal and is dilated more than 5 to 6 cm in diameter. This may happen remotely from the initial repair. Other indications for surgery include persistence of the dissection after the initial repair, recurrence of dissection, and infected prosthesis. The proximal aorta is the most common site requiring repeat surgery. The incidence of new dissection is 0.03% to 0.10%.[7] The current guidelines regarding the follow-up of patients after aortic dissection are summarized in **Table 4**.

Asymptomatic aortic aneurysms

Asymptomatic abdominal aortic aneurysms (AAA) are of concern because they put patients at risk of progressive dilation and rupture, compression of adjacent structures, and embolism of mural thrombus.[18] They often remain undetected until they are at imminent risk of rupture. For unknown reasons, male sex and smoking are the strongest risk factors for their development. The most important predictor of complications from AAA remains the size of the aneurysm. In 2005 the American Heart Association and the American College of Cardiology published guidelines for screening, surveillance, and repair, as shown in **Table 5**.[18] Currently, screening with physical examination and abdominal ultrasonography is recommended for men older than 60 who have parents or siblings who have ever been diagnosed with AAA (Class I). Screening is also recommended for men aged 65

Table 5. AHA guidelines for abdominal aortic aneurysm (AAA) screening, surveillance, and repair.

Intervention	Recommendations	Level of evidence
Screening	• Men ≥ 60 years who are either the siblings or offspring of patients with AAA should undergo physical examination and screening abdominal ultrasound.	Class I
	• Men 65–75 years who have ever smoked should undergo physical examination and screening abdominal ultrasound.	Class IIa
Surveillance	• Patients with AAA 4 cm to 5.4 cm in diameter should be monitored by ultrasound or CT scans every 6 to 12 months to detect expansion.	Class I
	• Patients with AAA < 4.0 cm in diameter should be monitored by ultrasound every 2–3 years.	Class IIa
Repair	• Patients with infrarenal or juxtarenal AAA > 5.4 cm in diameter should undergo repair.	Class I
	• Repair can be beneficial in patients with infrarenal or juxtarenal AAA measuring 5.0–5.4 cm in diameter.	Class IIa
	• Repair may be indicated in patients with suprarenal or thoracoabdominal AAA measuring 5.5–6.0 cm in diameter.	Class IIa

to 75 who have any smoking history (Class IIa).

When aneurysms are found, current guidelines require following any measuring 4.0 to 5.4 cm in diameter every 6 to 12 months by either abdominal ultrasound or CT. Patients with aneurysms measuring 5.5 cm or greater should undergo repair. For symptomatic AAA, repair is indicated regardless of diameter.

When the 2005 AHA guidelines were published, no randomized controlled trials had been carried out to assess the appropriate size at which to intervene on aneurysms involving the thoracic aorta. This remains the case today. Given the increased morbidity associated with thoracic aorta repair, the AHA guidelines recommend a slightly greater diameter (5.5 to 6.0 cm) for repair of suprarenal or thoracoabdominal aneurysms. However, the IRAD registry has shown aneurysm size to be a poor predictor of risk of dissection, as the majority of dissections occur at diameters less than 5.5 cm.[19]

Conclusions

Aortic dissection and ruptured abdominal aortic aneurysm are chief among life-threatening aortic conditions presenting to the emergency department. As the symptoms of these are often nonspecific, physicians need to have a high index of suspicion and to be aware of strategies for the diagnosis and management of arterial pathology. Through the development of innovative surgical techniques such as cardiopulmonary bypass and synthetic grafts, a uniformly fatal diagnosis has been turned into one where survival is possible if appropriate management is instituted in a timely manner. Much of the credit for this change rests with the pioneering work of surgeon Michael Ellis DeBakey.

Competing interests

None declared.

References

1. Meszaros I, Morocz J, Szlavi J, et al. Epidemiology and clinicopathology of aortic dissection. Chest 2000;117:1271-1278.
2. Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): New insights into an old disease. JAMA 2000;283:897-903.
3. Golledge J, Eagle KA. Acute aortic dissection. Lancet 2008;372(9632):55-66.
4. Libby P, Bonow R, Mann D, et al. Braunwald's heart disease. 8th ed. Philadelphia: Saunders/Elsevier; 2008:989.
5. Nienaber CA, Eagle KA. Aortic dissection: New frontiers in diagnosis and management: Part I: From etiology to diagnostic strategies. Circulation 2003;108:623-635.
6. Ince H, Nienaber CA. Diagnosis and management of patients with aortic dissection. Heart 2007;93:266-270.
7. Erbel R, Alfonso F, Boileau C, et al. Diagnosis and management of aortic dissection. Recommendations on the management of aortic dissection, European Society of Cardiology. Eur Heart J 2001;22:1642-1681.
8. Ramanath VS, Oh JK, Sundt TM, et al. Acute aortic syndromes and thoracic aortic aneurysm. Mayo Clin Proc 2009;84:465-481.
9. Spittell PC, Spittell JA Jr, Joyce JW, et al. Clinical features and differential diagnosis of aortic dissection: Experience with 236 cases (1980 through 1990). Mayo Clin Proc 1993;68:642-651.
10. Sommer T, Fehske W, Holzknecht N, et al. Aortic dissection: A comparative study of diagnosis with spiral CT, multiplanar transesophageal echocardiography, and MR imaging. Radiology 1996;199:347-352.
11. Nienaber CA, von Kodolitsch Y, Nicolas V, et al. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. N Engl J Med 1993;328:1-9.
12. Nienaber CA, Eagle KA. Aortic dissection: New frontiers in diagnosis and management: Part II: Therapeutic management and follow-up. Circulation 2003;108:772-778.
13. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003;42:1206.
14. Chiappini B, Schepens M, Tan E, et al. Early and late outcomes of acute type A aortic dissection: Analysis of risk factors in 487 consecutive patients. Eur Heart J 2005;26:180-186.
15. Suzuki T, Mehta RH, Ince H, et al. Clinical profiles and outcomes of acute type B aortic dissection in the current era: Lessons from the International Registry of Aortic Dissection (IRAD). Circulation 2003;108 (suppl 1):II312-317.
16. Umana JP, Lai DT, Mitchell RS, et al. Is medical therapy still the optimal treatment strategy for patients with acute type B aortic dissections? J Thorac Cardiovasc Surg 2002;124:896-910.
17. Maron BJ, Chaitman BR, Ackerman MJ, et al. Recommendations for physical activity and recreational sports participation for young patients with genetic cardiovascular diseases. Circulation 2004;109:2807-2816.
18. ACC/AHA 2005 Practice Guidelines for the management of patients with peripheral arterial diease (lower extremity, renal, mesenteric, and abdominal aortic). Circulation 2006;113:1474-1547.
19. Pape LA, Tsai TT, Isselbacher EM, et al. Aortic diameter > or = 5.5 cm is not a good predictor of type A aortic dissection: Observations from the International Registry of Acute Aortic Dissection (IRAD). Circulation 2007;116:1120-1127.

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