Spontaneous Aortic Dissection in the Presence of Coexistent or Previously Repaired Atherosclerotic Aortic Aneurysm

RICHARD P. CAMBRIA, M.D., DAVID C. BREWSTER, M.D., ASHBY C. MONCURE, M.D., FRED L. STEINBERG, M.D., and WILLIAM M. ABBOTT, M.D.

Acute aortic dissection occurred in 18 patients who had previously diagnosed atherosclerotic aneurysms of the thoracic and/or abdominal aorta. These patients were reviewed to assess the clinical course when these two forms of aortic pathology coexist. Patients were grouped according to status of their atherosclerotic aneurysm (previously repaired *vs.* untreated) and the segments of the aorta effected by the acute spontaneous dissection. Group 1 patients (n = 5) had previously undergone-abdominal aortic aneurysmectomy (AAA) repair, and the abdominal aortic suture line effectively terminated the dissection process. In Group 2 patients (n = 5), the acute dissection and the atherosclerotic aneurysm involved different segments of the aorta. Group 3 patients (n = 8) experienced spontaneous aortic dissection involving atherosclerotic aneurysms (five infrarenal, three thoracoabdominal), with threatened or actual rupture occurring in six patients, resulting in three deaths. In Group 3 patients, rupture occurred both at the atherosclerotic aneurysm (four patients) and at the site of the aortic intimal tear of the dissection (two patients) after AAA repair. The use of Magnetic Resonance Imaging (MRI) has proven to be highly accurate in delineating the nature and extent of pathology in recently encountered patients with complicated aortic disease. Coexistence of atherosclerotic aneurysm and acute dissection appears to increase the risk of aortic rupture, in both proximal and distal aortic segments.

From the Vascular Surgery and Radiology Services, Massachusetts General Hospital, and the Departments of Surgery and Radiology, Harvard Medical School, Boston, Massachusetts

RUPTURE OF THE AORTA occurs in nearly 20% of all patients with acute aortic dissection, and this complication is strongly correlated with early mortality.[1,2] Rupture complicating spontaneous aortic dissection generally occurs in the proximal thoracic aorta, at the site of the aortic intimal tear.[3,4] Patients with preexistent atherosclerotic aneurysm of the thoracic and/or abdominal aorta and patients who had undergone previous prior graft repair of the thoracic and/or abdominal aorta harbor a locus in the aorta that may potentially change both the incidence and site of rupture. As such, indications for definitive surgical therapy in this relatively uncommon circumstance may differ from the accepted approach for both the atherosclerotic aneurysm and the acute dissection. This issue is particularly relevant in that population where these two types of pathology are likely to coexist—that is, in patients with Debakey Type III dissections (originating in the descending thoracic aorta), who tend to be older and who have a higher incidence of hypertension when compared with those having Types I and II dissection.[1,2] Furthermore, the role of surgical therapy in these patients requires definition because treatment of patients with uncomplicated Type III dissections may ordinarily consist of medical therapy alone.[5]

We reviewed the course of a group of patients afflicted with both atherosclerotic aortic aneursym and acute dissection of the aorta to determine if their coexistence altered the expected natural history of either lesion or the optimal mode and priority of treatment. We also report recent experience with Magnetic Resonance Imaging (MRI) in the evaluation of these patients.

Patients and Results

Patients were identified from recent experience and from a previous review of over 300 patients with aortic dissection.[1] In all cases, the diagnosis of both atherosclerotic (the terms "atherosclerotic" and "degenerative" are used interchangeably) aneurysm and aortic dissection were made by a combination of clinical, radiographic, and operative or autopsy findings. Spontaneous dissections only were considered. Aortic dissection was classified according to the Debakey system,[2] and the acute phase of the disease was defined as the 2-week period after the onset of symp-

Reprint requests and correspondence: Richard P. Cambria, M.D., 15 Parkman Street, ACC 358, Boston, MA 02114.

Submitted for publication: March 3, 1988.

TABLE 1. *Clinical Characteristics of Group 3 Patients*

Patient No.	Type AD	Size/Location of Athero Aneurysm	Location/Date of Rupture	Therapy	Outcome
1	III B	7 cm/AAA	Thoracic Intimal Tear—7/71	AAA graft TA graft	Died secondary to TA rupture
2	III B	8 cm/AAA	AAA—2/76	Medical for AD AAA graft	Died secondary to AAA rupture
3	III B	7 cm/AAA	Thoracic Intimal Tear—12/75	AAA graft	Died secondary to TA rupture, 13th postop day after AAA graft
4	III B	6 cm/AAA	Junction AAA and AD—9/83	TAA graft	Did well
5	III A	5 cm/TAA	Junction TAA and AD—9/84	TAA graft	Intraoperative death
6	III B	6 cm/AAA	Junction AAA and AD—10/85	AAA graft	Died of renal failure after ruptured AAA
7	III A*	5 cm/TAA	None	AAA graft TAA graft	Did well
8	III B	5 cm/TAA	None	Medical RX AD Urgent CABG	Awaiting aortic repair

* Atypical dissection originated in TAA.
AAA = Abdominal Aortic Aneurysm.
TA = Thoracic Aorta.
TAA = Thoracoabdominal Aortic Aneurysm.
AD = Acute Dissection.
Athero Aneurysm = atherosclerotic aneurysm.

toms. In our hospital, Types I and II dissections are treated with prompt surgical repair, whereas uncomplicated Type III dissections are preferentially treated with medical therapy. The status of the atherosclerotic aneurysm (untreated *vs.* previously grafted) and whether or not the dissection process and degenerative aneurysm involved the same segments of the aorta permitted convenient division of patients into three groups.

Group 1

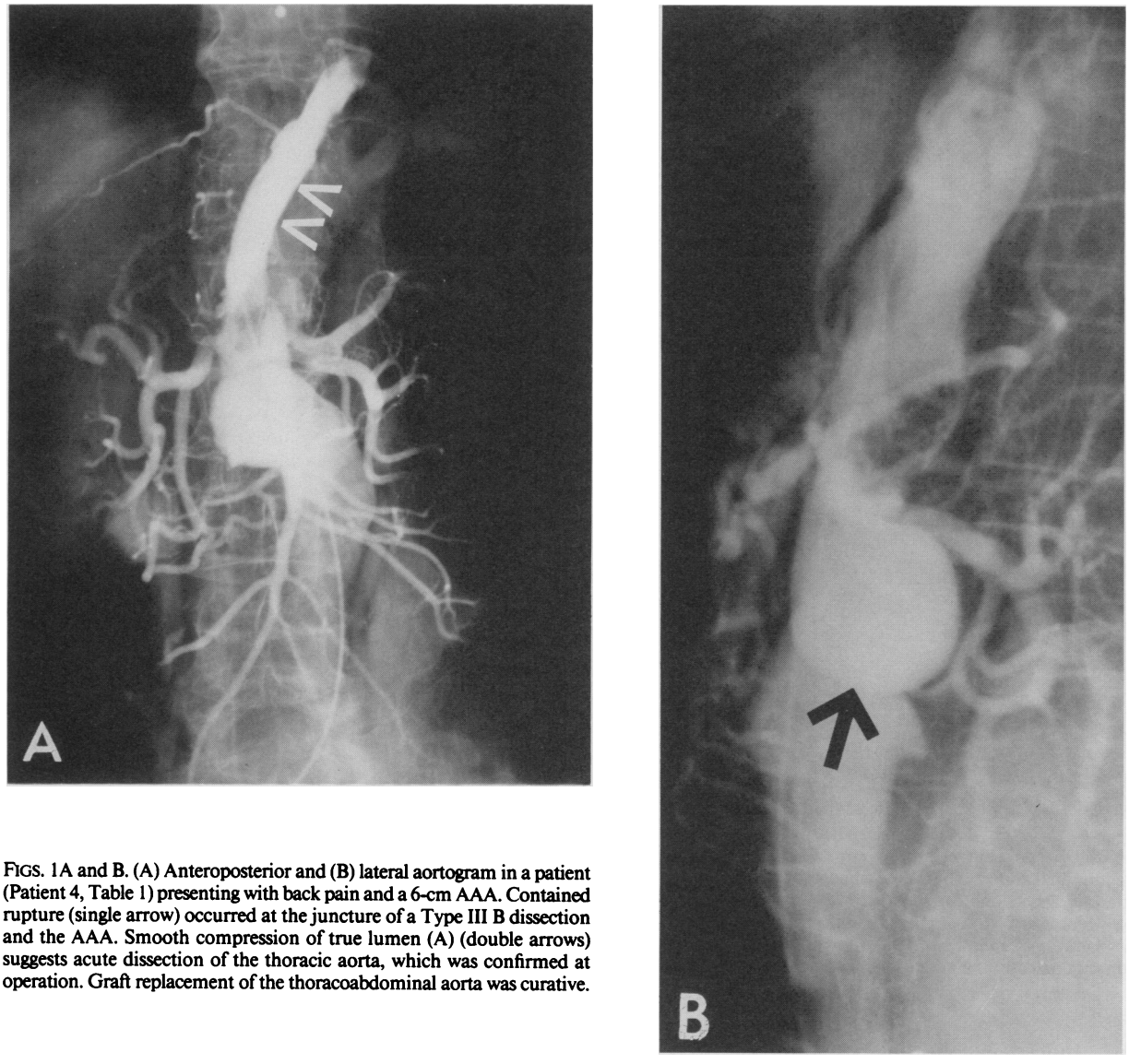
The five patients of Group 1 had undergone elective grafting of an infrarenal abdominal aortic aneurysm (AAA) before presenting with a spontaneous aortic dissection that progressed to the abdominal aorta and involved the site of previous aortic surgery. The longest interval between AAA repair and spontaneous dissection was 7 years, but for five patients, this interval was less than 3 years. All of these patients sustained Type III B dissections (intimal tear distal to the left subclavian artery and dissection proceeding through the abdominal aorta), which terminated at the suture line of the previous AAA repair, as verified by angiography (in two patients), operative exploration (in two patients) and autopsy (in one patient). A single patient experienced rupture of the thoracic aorta (at the site of the intimal tear) and died, whereas two additional patients died of renal failure in the acute phase of the dissection. Rupture at a previous aortic suture line was not seen.

Group 2

The five patients of Group 2 experienced acute dissections; one patient experienced Type I, two patients had Type II, and two patients had Type III A. These acute dissections did not involve the infrarenal aorta, where three patients had untreated AAA and two additional patients had previously undergone AAA resection. Thus, the spontaneous dissection and the degenerative aneurysm involved different segments of the aorta. A single patient died during attempted correction of a Type I dissection. Two survivors of definitive surgical repair of Type II dissection underwent subsequent AAA repair within 1 year with good results. The two patients who had Type III A (confined to the descending thoracic aorta) dissections and who had had previous AAA repair were treated medically with favorable outcomes.

Group 3

The eight patients of Group 3 experienced acute dissection that extended to or involved coexisting unoperated atherosclerotic aneurysms (five patients had AAA, and three had thoracoabdominal aneurysm). These patients harbored Type III dissections for either days or weeks, and thoracic aortic rupture (at the site of the intimal tear) occurred in two patients after graft repair of the atherosclerotic AAA. In four additional patients (Patients 2, 4, 5, and 6—Table 1), contained or frank rupture occurred

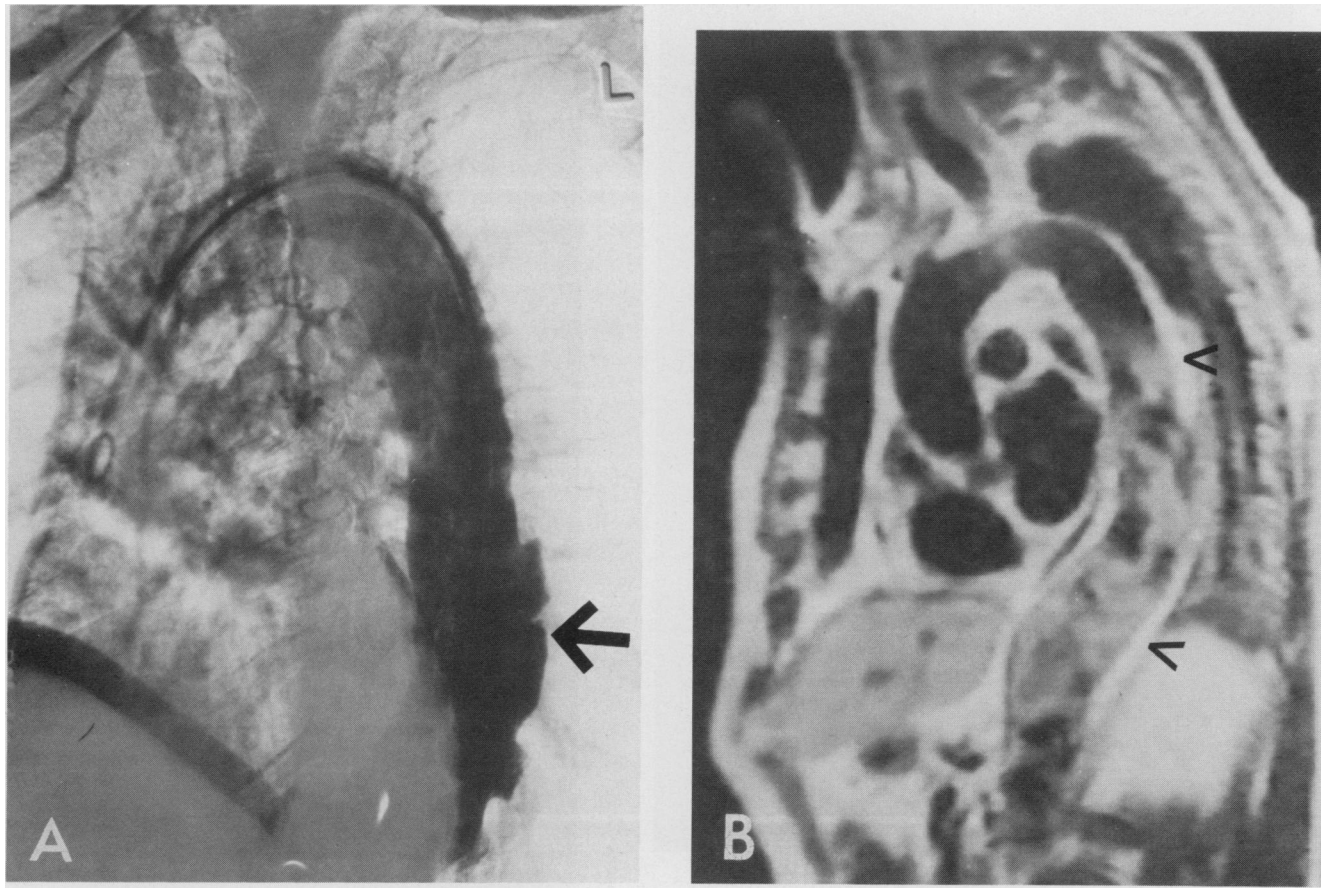


FIGS. 1A and B. (A) Anteroposterior and (B) lateral aortogram in a patient (Patient 4, Table 1) presenting with back pain and a 6-cm AAA. Contained rupture (single arrow) occurred at the juncture of a Type III B dissection and the AAA. Smooth compression of true lumen (A) (double arrows) suggests acute dissection of the thoracic aorta, which was confirmed at operation. Graft replacement of the thoracoabdominal aorta was curative.

at the junction of the dissecting process and the atherosclerotic aneurysm (Figs. 1A and B). In six patients presenting with back pain and known degenerative aneurysms, the presence of aortic dissection was not appreciated on the basis of clinical and angiographic findings. This diagnostic uncertainty contributed to one death (Patient 5, Table 1) when technical difficulties in dealing with the dissected aorta arose. Recent experience with two patients (Patients 7 and 8, Table 1) who presented with back pain and extensive atherosclerotic aneurysm of the thoracoabdominal aorta has demonstrated the utility of MRI in such circumstances. Both patients were shown to have superimposed Type III B dissections to account for their symptoms of back pain. The diagnosis of the first patient was based on the detection of an intimal flap and two patent vascular lumens in the descending thoracic aorta. In the second patient, the diagnosis was made by the detection of hematoma in the wall of the thoracoabdominal aorta. The angiographic and computed tomographic (CT) images were unable to distinguish dissection from irregular thrombus in an aortic aneurysm. In Patient 7, operative findings confirmed the precise extent and nature of pathology suggested by MRI (Figs. 2A and B).

Discussion

Acute dissection occurring in an aorta previously afflicted with degenerative, atherosclerotic aneurysm is unusual. Large autopsy and clinical series of aortic dissection make scant mention of this combination of aortic pa-



FIGS. 2A and B. (A) Thoracic aortogram and (B) MRI scan of a patient (Patient 7, Table 1) presenting with back pain and an aneurysm of the thoracoabdominal aorta as defined by angiography and CT scan. Angiography revealed irregular atherosclerotic plaque (single arrow) but no definitive signs of aortic dissection. T1 weighted MRI scan demonstrated high intensity (bright) linear regions in the outer circumference of the aorta (B) (arrows), which was also seen on T2 weighted images. These were interpreted as early (fresh) hematoma, thus indicating a dissection. Note also irregular plaque corresponding to that shown on aortogram (A). Operative findings confirmed the radiographic diagnosis and demonstrated that the dissection originated in the atherosclerotic aneurysm. Graft replacement of the thoracoabdominal aorta was curative.

thology.[6,7] Indeed, in collecting both our recent experience and patients identified in a 20-year review of 325 cases of spontaneous dissection, it appears that only 5% of acute dissections occur in the aorta of patients with coexistent or previously operated degenerative aneurysmal disease.[1] This is due, in part, to the lower mean age of those afflicted with acute dissection.[2] However, in our experience, the mean age of patients with distal (Type III A and B) dissections (63 years) is not significantly different from that of our elective AAA surgery population (69 years). Thus, the rare coexistence of these forms of aortic pathology is consistent with the prevailing opinion concerning their different etiologic factors.[3,4]

Not surprisingly, the combination of degenerative aneurysm and acute dissection increased the risk of aortic rupture. Three fourths of our Group 3 patients experienced rupture (four within the degenerative aneurysm and two at the site of the aortic intimal tear). All of these patients had Type III dissections, for which the risk of rupture is ordinarily in the range of 10%, a percentage significantly less than that of the risk of rupture in patients with proximal (Types I and II) dissections.[1] This difference in the risk of rupture forms, in part, the rationale for medical therapy in patients with uncomplicated Type III dissections.[5] The frequency of rupture of the abdominal aorta in the patients detailed in this report is quite atypical of patients who have had acute dissection. Although rupture in the abdominal aorta has been recognized since the early days of surgical therapy for dissection,[8] and Hunter et al. have demonstrated the utility of abdominal aortic graft replacement in this setting, such abdominal aortic rupture is rare.[9] In our review of 325 patients with aortic dissection, rupture in the abdomen occurred only in the setting of antecedent degenerative aneurysm.[1]

These findings have important implications for therapy in an era when "uncomplicated" Types III A and B dissection are preferentially treated with medical therapy in many institutions, including our own. Our experience would indicate that the presence of a juxtaposed atherosclerotic aneurysm greater than 5 cm constitutes a "complicated" Type III dissection, and standard antihypertensive therapy has failed to prevent aortic rupture. Because rupture occurred most frequently in the degenerative aneurysm, initial surgical priority should be given to the aortic segment where both forms of pathology coexist (usually the infrarenal abdominal aorta). However, if abdominal operation is conducted during the acute phase of the dissection, the proximal aortic suture line should be fenestrated, as previously emphasized, to forestall rupture at the uncorrected proximal intimal tear.[1,9,10] Two of our Group 3 patients experienced thoracic rupture of their dissections, at 2 and 8 weeks after abdominal aneurysm repair, respectively. In both of these patients, significant aneurysmal dilatation of the outer wall of the false lumen in the chest was present, indicating risk of rupture and the need for definitive surgical repair of the origin of the dissection.

The role of atherosclerotic plaque in the natural history of aortic dissection is uncertain. The careful analysis of pathology made by Roberts suggests that atherosclerotic plaque frequently serves to terminate the dissection process.[4] Thus, in younger patients with a proximal intimal tear, the entire aorta is often involved (Type I dissection). Yet, after reviewing 64 surgically treated patients with Types III A and B dissections, Jex et al. noted atherosclerosis in 83% of these patients. Slater and DeSanctis reported that dissection can originate in atherosclerotic plaque, and this was confirmed at the time of operation in one of our patients.[11] Our findings suggest that, although atherosclerotic plaque itself may be "protective" in serving to terminate the dissection process, the situation is quite different when atherosclerotic or degenerative aneurysm is present. In such a circumstance, rupture of the preexistent aneurysm is the more likely scenario.

MRI was particularly useful in the case of two patients (Patients 7 and 8) who were recently seen with known thoracoabdominal aneurysm and who presented with back pain (Table 1, Fig. 2A and B). MRI is a powerful imaging modality for investigating the vascular tree because of its multidimensional display capabilities, the special effects of flowing blood, and the high contrast between flowing blood and the vascular wall. MRI offers a number of advantages for tissue characterization of the wall of the aorta. Specifically, T1 and T2 weighted images have the ability to identify a perivascular or intramural hematoma, and characterize the components present in the blood clot.[12] The natural evolution of a hematoma may be followed, based on MRI analysis of its chemical constituents. These chemical constituents have different magnetic and paramagnetic properties that alter the signal intensity on the magnetic resonance image. Increased signal on a T1 weighted image with an increased signal on a T2 weighted image when methemoglobin is present in the hematoma has been described.[12] Although slow flow in the lumen of the aorta may also show bright signal on certain pulse sequences, hematoma in the wall of the aorta can be distinguished by a combination of T1 and T2 weighted pulse sequences.[13] The definitive MR diagnosis of aortic dissection requires identification of an intimal flap, with blood flow on each side of the flap when the false channel is patent.[14] In the case where the false channel is thrombosed, MRI appears useful in distinguishing fresh clot from chronic mural thrombus.[12] Another advantage of MRI is its ability to image aneurysms and dissections with respect to the great vessels of the aortic arch, mesenteric, and renal arteries. Although the CT scan is generally able to exclude aneurysm rupture, both the CT scan and angiography failed to delineate the presence of aortic dissection in several of these patients. This was useful information, affording an explanation for the back pain (dissection *vs.* rupture) and permitting deliberate, elective operation to be performed. In one case (Patient 7, Table 1) the dissection was found to originate within a thoracoabdominal aneurysm in the region of the diaphragm and to extend in retrograde fashion and thus involve the distal half of the descending aorta. No threat of an uncorrected proximal intimal tear remained. In another patient with an atherosclerotic TAA (Patient 8, Table 1), MRI detected a dissection channel that accounted for back pain. Unstable angina prompted coronary revascularization, and the aorta was treated with medical therapy. Interval graft replacement of the aneurysm is planned.

Summary

The unusual coexistence of aortic dissection and a preexistent atherosclerotic aneurysm appears to increase the risk of rupture, particularly at the confluence of the two lesions. When these two forms of aortic disease occur synchronously, but do not involve the same segments of the aorta, the natural history appears to conform to the expected course of each lesion considered individually. An aortic suture line from a previously placed aortic graft appears protective in terminating a subsequent acute dissection. MRI has proven useful and accurate in delineating the nature and extent of pathology in these patients. The prohibitive risk of rupture in such patients warrants an aggressive surgical approach.

References

1. Cambria RP, Brewster DC, Gertler J, et al. Vascular complications associated with spontaneous aortic dissection. J Vasc Surg 1988; 7:199–209.
2. DeBakey ME, McCollun CH, Crawford ES, et al. Dissection and dissecting aneurysms of the aorta: twenty-year follow-up of five hundred twenty-seven patients treated surgically. Surgery 1982; 92:1118–1133.
3. Crawford ES, Crawford JL. Aortic dissection and dissecting aortic aneurysm. *In* Diseases of the Aorta. Baltimore: Williams and Wilkins, 1984; 168–173.
4. Roberts W. Aortic dissection: anatomy, consequences, and causes. Am Heart J 1981; 10:195–214.
5. DeSanctis RW, Doroghazi RM, Austen WG, Buckley MJ. Aortic dissection. N Engl J Med 1987; 317:1060–1067.
6. Hirst AE, Johns VJ, Kime SW. Dissecting aneurysm of the aorta: a review of 505 cases. Medicine 1958; 37:217–279.
7. Miller DC, Mitchell RS, Oyer PE, et al. Independent determinates of operative mortality for patients with aortic dissections. Circulation 1984; 70:153–164.
8. Johns T. Dissecting aneurysm of the abdominal aorta. Ann Surg 1953; 137:232–235.
9. Hunter JA, Dye WS, Javid H, et al. Abdominal aortic resection in thoracic dissection. Arch Surg 1976; 111:1258–1262.
10. Cambria RP, Morse S, August D, Gusberg R. Acute dissection originating in the abdominal aorta. J Vasc Surg 1987; 5:495–497.
11. Slater EE, DeSanctis RW. The clinical recognition of dissecting aortic aneurysm. Am J Med. 1976; 60:625–633.
12. Gomori JM, Grossman RI, Goldberg HI, et al. Intracranial hematomas, imaging by high field MR. Radiology 1985; 157:87–93.
13. Von Schultess GK, Higgins CB. Blood flow imaging with MR: spin phase phenomena. Radiology 1984; 157:687–690.
14. Amparo EG, Higgins CB, Hrick L, Sollito R. Aortic dissection: magnetic resonance imaging. Radiology 1985; 155:399–406.