Aortic Dissection with the Entrance Tear in the Descending Thoracic Aorta

Analysis of 40 Necropsy Patients

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Clinical and necropsy findings are described in 40 patients who had aortic dissection with the entrance tear in the descending thoracic aorta. Their ages at death ranged from 39 to 91 years (mean, 66 years); 24 (60%) were men and 16 (40%) were women. Systemic hypertension was present by history in 33 patients (83%) and the hearts were of increased weight in 78%. Of the 40 patients, 31 (78%) had no operative intervention, while 9 (22%) underwent operation for aortic dissection. Of the 31 patients without operative therapy, the diagnosis of aortic dissection was established in life in 9 patients (29%) and at necropsy in 22 (71%). The interval from aortic dissection to death was 30 days or less in 13 patients (42%); rupture of the false channel was the cause of death in 9 patients (69%), renal failure in 2 (15%), and the cause was unclear in 2 (15%). The interval from aortic dissection to death was more than 30 days in 18 (58%) of the 31 patients without operative therapy. The cause of death in these 18 patients was related to the dissection in 11 (61%) (rupture of the false channel in 5; renal failure from dissection in 3, and rupture of the false channel of a second acute dissection in 3), but in the other 7 patients (39%) death was unrelated to the dissection but a nonfatal complication, specifically stenosis of the true channel from compression by a thrombus-filled false channel, occurred in 4 of these 7 patients. Thus only 3 (10%) of the 31 patients without operative therapy had no complications of aortic dissection. All nine patients who underwent operation had had an aortic dissection within 30 days, and the operation was performed because of a major complication of the dissection. Four patients survived 8 to 84 months after the operation. Thus early operative intervention (before the appearance of complications) appears justified in patients with aortic dissection with the entrance tear in the descending thoracic aorta to prevent rupture of the false channel acutely or after initial healing; to prevent renal failure from compression of renal arteries by an aneurysmal false channel; to prevent true channel stenosis from compression by a thrombus-filled false channel; and possibly to prevent the recurrence of acute dissection.

DURING THE PAST 30 years, 182 patients with spontaneous (noniatrogenic) aortic dissection were studied at necropsy in the Pathology Branch, National Heart, Lung, and Blood Institute. The entrance tear was located in the ascending aorta in 128 patients (70%), transverse aorta in 12 patients (7%), descending thoracic aorta in 40 patients (22%), and abdominal aorta in 2 patients (1%). This report describes certain clinical and necropsy findings in the 40 patients in whom the entrance tear was located in the descending thoracic aorta and discusses therapeutic implications of the findings.

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Methods

The necropsy records of the Pathology Branch, National Heart, Lung, and Blood Institute from 1959 to April 1990 were searched for cases coded as 'aortic dissection.' A total of 194 cases were so coded. Twelve were excluded because of an iatrogenic etiology—the aortic entrance tear was adjacent to an aortotomy. All 142 cases with the entrance tear in either ascending, transverse, or abdominal aorta also were excluded from this analysis. The remaining 40 cases had the entrance tear in the descending thoracic aorta and they constitute the study group.

The hearts and aortas in all 40 patients originally were examined and classified by W. C. Roberts; 26 of the 40 cases were examined by C. S. Roberts and re-examined by W. C. Roberts. Of the 14 cases not re-examined, photographs of the aortas were available for examination in seven. Of the 40 patients, 1 was studied at necropsy in 1968, 20 were studied from 1971 to 1980, and 19 from 1981 to 1990. The 40 cases were submitted from 11 different hospitals: 38 cases from local and 2 cases from nonlocal hospitals. Of the 40 cases, 1 hospital submitted 11 cases; 1 hospital, 8 cases; 2 hospitals, 4 cases each; 2 hospitals, 3 cases each; 3 hospitals, 2 cases each; and 3

TABLE 1. *Clinical and Morphologic Observations in 31 Necropsy Patients with Aortic Dissection with the Entrance Tear in Descending Thoracic Aorta*

Patient Number	Age at Death (Years)	Sex	SH	Aorto-gram	AD Diagnosed Clinically	Interval AD to Death	AR	Mode of Death RF	Other	Aortic Dissection A	Aortic Dissection H	Dissection in Abdominal Aorta	FC Thrombus	FC Rupture	TC Stenosis	Reentry Tear	AAA	HW (g)	LV Fibrosis
1	40	F	+	0	0	7d	+	0	0	+	0	+(a)	+	+	0	0	0	—	0
2	49	F	+	0	+	30d	+	0	0	+	0	0	+	+	0	0	0	360	0
3	57	M	+	0	0	1d	+	0	0	+	0	++	+	+	0	0	0	360	0
4	58	M	+	0	0	9d	+	0	0	+	0	0	++	+	+(b)	0	0	810	0
5	60	M	0	0	0	4d	+	0	0	+	0	+	+	+	0	0	0	340	0
6	63	F	+	0	0	7d	0	0	U	+	0	0(c)	+	0	0	0	0	590	0
7	64	M	+	+	+	7d	0	0	U	+	0	++	++	0	++(b)	0	0	—	0
8	65	M	0	+	+	10d	0	+	0	+	0	+	++	0	+	0	0	495	0
9	65	F	0	+	0	14d	+	0	0	+	0	+	+	+	0	0	0	—	+
10	69	F	+	0	0	28d	+	0	0	+	0	++(c)	+	+	0	0	0	435	0
11	71	M	+	+	+	21d	0	+	0	+	0	0	++	0	+	0	+(d)	560	+
12	76	F	+	0	0	14d	+	0	0	+	0	0	+	+	0	0	0	—	0
13	84	M	+	0	0	9d	+	0	0	+	0	+	++	+	0	0	0	510	0
14	58	M	+	+	0	—	0	0	C	0	+	++	++	0	++	+(FA)	0	—	+
15	61	M	+	0	+	19m	+	0	0	0	+	+	0	+	0	0	+	400	0
16	63	M	+	0	+	72m	+	0	0	0	+	++	+	+	+	0	0	450	+
17	63	M	+	0	0	—	0	0	CAD(h)	0	+	+	++	0	++	0	0	—	0(i)
18	66	F	+	0	+	2m	+	0	0	0	+	+	++	+	+	+	0	—	0
19	66	M	+	0	0	35m	0	0	MS	0	+	0	0	0	0	+	0	900	0
20	67	M	+	0	0	—	+	0	0	0	+	++	+	+	+	+	0	400	0
21	67	M	+	0	+	72m	+(f)	0	0	0	+	+	++	0	++	0	+	—	+
22	69	F	+	0	0	—	0	+	0	0	+	++	+	0	0	0	0	—	0
23	69	M	0	0	0	—	0	0	Cancer	0	+	+	+	0	0	0	0	330	0
24	76	F	+	0	0	—	0	+	0	0	+	+	+	0	++	+	0	610	0
25	78	M	0	0	0	—	0	0	CAD(g)	0	+	++	+	0	+	+	0	300	0
26	80	F	0	0	0	96m	0	+	0	0	+	++	+	0	++	+(CIA)	0	400	0
27	83	M	+	0	0	—	0	0	CAD	0	+	0	++	0	0	0	+	530	+
28	91	F	+	0	0	—	+	0	0	0	+	0	+	+	0	+	0	380	0
29	48	M	+	+(e)	0	—	+(e)	0	0	+e	+	+	0	0	0	0	0	440	0
30	52	M	+	+(e)	+	60m	+(e)	0	0	+e	+	+	+	0	++	+	0	500	0
31	63	M	+	+(e)	0	7m	+(e)	0	0	+e	+	0	++	0	+	0	+	550	+
Totals or Means	66	20M, 11F	25	13	9		18	5	8	16	18	22	28	14	15	9	5		7
(%)			81	42	29		58	16	26	52	58	71	90	45	48		16		23

A, acute; AAA, abdominal aortic aneurysm, fusiform, atherosclerotic origin; AD, aortic dissection; AR, aortic rupture; C, cirrhosis, alcoholic type; CIA, common iliac artery; d, days; F, female; FA, femoral artery; FC, false channel; H, healed; HW, heart weight; LV, left ventricular; M, male; m, months; MS, Marfan syndrome; RF, renal failure; TC, true channel; SH, systemic hypertension; u, uncertain; CAD, coronary artery dissection.

(a) One plus means the dissection involves only a portion of abdominal aorta; 2+ involves all the abdominal aorta.

(b) One plus indicates relatively small or mild, and 2+, relatively large or severe.

(c) Retrograde and antegrade aortic dissection.

(d) Earlier operative resection of the abdominal aortic aneurysm.

(e) Acute aortic dissection ruptured, not the healed one.

(f) Fusiform atherosclerotic abdominal aortic aneurysm ruptured, not the aortic dissection.

(g) Sudden coronary death outside the hospital.

(h) Acute myocardial infarction with fatal rupture of the left ventricular free wall.

(i) This was the only patient with an acute myocardial infarct.

TABLE 2. *Clinical and Morphologic Characteristics in Nine Patients Who Underwent Operative Therapy for Acute Aortic Dissection with the Entrance Tear in the Descending Thoracic Aorta*

Case	Age at Death (years)	Sex	SH (Hx)	Indication for Operation			Interval AD to Operation (days)	Operation		Interval First Operation to Death	Mode of Death			Entry Tear Upper 1/2	Involve Abdominal Aorta	Retrograde AD	Reentry Tear	HW (g)	LV Fibrosis
				H	Organ Ischemia	Other		Graft	Plication		Reop	H	I	Other						
1	66	M	+	+	0	0	6	+	0	0	0	+	0	0	+	+	0	0	495	0
2	39	F	+	0	+	0	1	0	+	<1 days	0	0	0	RF	+	+	0	0	460	0
3	69	F	+	0	+	0	4	0	0*	1 days	0	0	0	U	+	+	+	+	—	0
4	52	F	+	+	0	0	30	+	0	9 days	+	+	0	0	+	+	0	0	440	0
5	67	F	+	0	0	↑AD	9	+	0	9 days	0	+	0	0	+	+	0	0	295	+
6	65	F	+	—	—	—	≤30	++	0	8 mos	+	+	+	0	+	0	0	0	455	+
7	46	M	+	0	0	AR	≤30	+	0	10 mos	0	0	+	0	+	+	+	+	470	0
8	71	M	+	—	—	—	≤30	+	0	14 mos	0	+	+	0	+	+	0	0	400	0
9	53	M	0	0	+	0	≤30	+	0	84 mos	0	+	0	0	—	+	0	0	429	+
Totals and means	66	4M 5F	8 89%	2	3	2	≤30	7	1	—	2	6	3	2	8	8	2	2	—	3

AD, aortic dissection; AR, aortic rupture; CHF, congestive heart failure; D, days; F, female; g, grams; H, hemorrhage; Hx, history; HW, heart weight; I, infection; LV, left ventricle; M, male; m, months; Op, operation; Reop, reoperation; RF, renal failure; SH, systemic hypertension; U, uncertain.

* Fenestration procedure creating a passage between the true and false aortic channels.
† The graft was placed in ascending aorta and aortic valve replacement was performed for aortic regurgitation due to retrograde dissection.

hospitals, 1 case each. No case came from the Clinical Center of the National Institutes of Health.

Results

Pertinent clinical and necropsy findings in each of the 40 patients are summarized in Table 1 (31 without operative therapy) and Table 2 (9 with operative therapy). The ages at death of all 40 patients ranged from 39 to 91 years (mean, 66 years); 24 were men and 16 were women. Systemic hypertension was apparent by history in 33 patients (83%) and the hearts were increased in weight in 78% (more than 350 g in women; more than 400 g in men).

Nonoperative Cases

Certain clinical and necropsy findings in these 31 patients are summarized in Table 1 and illustrated in Figures 1 to 13. Their ages ranged from 40 to 91 years (mean, 66 years); 11 were women and 20 were men. Systemic hypertension had been present by history in 25 patients (81%), and the hearts were of increased weight in 82%. Of the 29 patients, only 9 (29%) had the diagnosis of aortic dissection established during life. Of the 13 patients who had an aortogram performed, diagnosis of suspected aortic dissection was confirmed in 9 patients.

Acute Dissection

In 13 patients (42%) (numbers 1 to 13; Table 1, Figs. 1 to 4), the aortic dissection at necropsy was acute. The interval from onset of signs and symptoms compatible with acute dissection varied from 4 to 30 days (mean 12 days, median 9 days). In only 4 of the 13 patients was aortic dissection diagnosed clinically. Death resulted from rupture of the outer wall of the false channel in 9 of the 13 patients, from acute renal failure in 2 patients, and from uncertain causes in 2 patients.

At necropsy the entrance tear was in the proximal half of the descending thoracic aortas in all 13 patients, and in each of the nine who ruptured, the external rupture site was in the vicinity of the entrance tear. The rupture in each of the nine patients caused extravasation of blood into the left pleural space. The dissection extended into the abdominal aorta in eight patients, involving all of it in only two patients. The false channels contained thrombus in all 13 patients, relatively small amounts in 8 patients, and it filled all or most of the false channel in 5 patients. In 4 patients, all of whom had thrombi filling most or all of the false channels, the lumen of the true channel was severely compressed, producing what might be called true aortic stenosis. A re-entry tear was not identified in any of the 13 patients. In two patients (numbers 5 and 9, Table 1), the aortic dissection progressed retrogradely, as well as antegradely, and the adventitial hem-

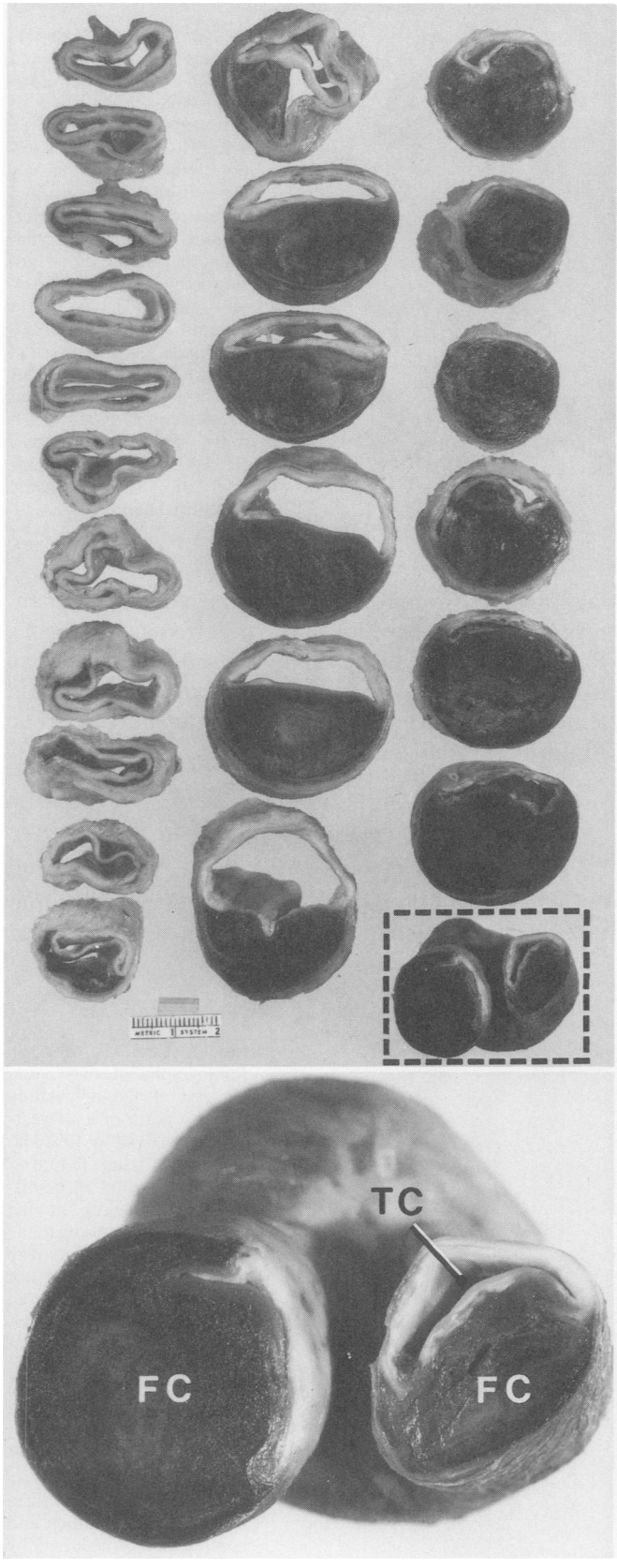


FIG. 1. (Case 7, Table 1). Serial cross-sections of aorta from the beginning of the descending thoracic aorta (top left) through the abdominal aortic bifurcation in a 64-year-old man (Howard #A79-40) who died of false channel rupture 7 days after onset of dissection with the entrance tear in the descending thoracic aorta. Thrombus occupies the entire false channel (FC) in the lower sections and compresses the true lumen, causing near-total obstruction of abdominal aorta. The close-up photograph (bottom) shows thrombus-filled false channels in each common iliac artery producing total true channel (TC) occlusion in one and partial true channel occlusion on the other.

orrhage was so extensive in one of these patients (number 9) that the mediastinum was widened, a finding that could have caused confusion with dissection from an entrance tear in the ascending aorta.[1-3] One patient (number 10) had an atherosclerotic fusiform aneurysm of the abdominal aorta that had been resected previously; the dissection, however, stopped 2 cm cephalad to the proximal graft suture line.

Healed Dissection

In 18 patients (58%), the dissection at necropsy had healed (numbers 14 to 31; Table 1, Figs. 5 to 13). The interval from onset of signs and symptoms compatible with acute dissection to death in the 7 patients in whom it was discernible ranged from 2 to 96 months (mean, 45 months; median, 35 months). Although 9 of the 18 patients had aortograms, in only 5 patients was aortic dissection diagnosed clinically. Death resulted from rupture of the healed false channel in 5 (28%) patients. Three additional patients (17%) (numbers 27, 28, and 29; Table 1) also died from rupture of the wall of the false channel, but the rupture site was the ascending aorta where a second acute new dissection had occurred. These three patients were the only ones who had both acute and healed aortic dissections. In each the entrance tear of the dissection that healed was the descending thoracic aorta, and the entrance tear for the fatal acute dissection was ascending aorta. Death in three patients (17%) resulted from chronic renal failure, which appeared to be the consequence of renal arterial compression by the large aneurysmal false channel. Of the remaining 7 patients, death was unrelated to the aortic dissection: 3 died from severe atherosclerotic coronary artery disease (1 suddenly, 1 from congestive heart failure, and 1 from left ventricular free wall rupture during acute myocardial infarction); 1 died from congestive heart failure, probably secondary to mitral regurgitation associated with the Marfan syndrome; 1 died of cancer; 1 of complications of alcoholic cirrhosis; and 1 of rupture of an atherosclerotic fusiform abdominal aortic aneurysm.

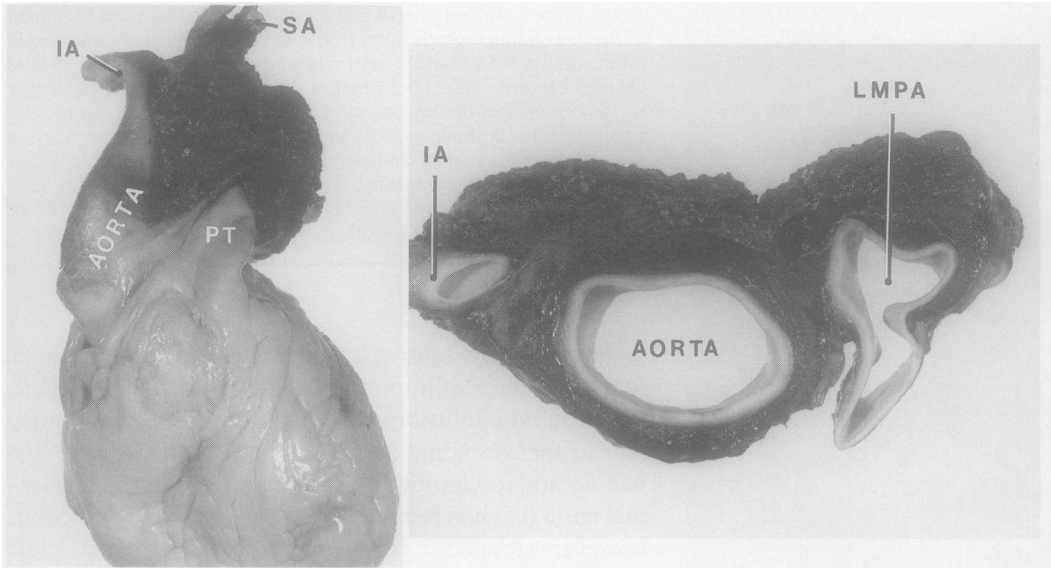


FIG. 2. (Case 10, Table 1) Photograph of heart and aorta of a 69-year-old woman (SH #A88-13) who had an entrance tear at the aortic isthmus with medial dissection to the common iliac arteries. In addition adventitial dissection occurred in a retrograde direction to ascending aorta and into the adventitia because both great arteries share a common adventitia. Left: Thrombus surrounds both aorta and pulmonary trunk (PT). SA = subclavian artery. Right: Cross-sections of aorta, left main pulmonary artery (LMPA), and innominate artery (IA) showing severe adventitial hemorrhage.

The entrance tear in 17 of the 18 patients was in the proximal half of the descending thoracic aorta. In the five patients with rupture of the wall of the 'healed' false channel, the rupture site was in the vicinity of the entrance tear. Of the patients with fatal rupture, the hemorrhage was into the left pleural space in four patients and into the esophagus in one (patient number 27) by a mechanism explained in Figure 11. The dissection involved the abdominal aorta in 14 patients with extension into 1 or both common iliac arteries in 6. The false channel contained thrombus in 15 of the 18 patients, and, in 6 of them, the thrombus occupied all or nearly all of the lumen of the false channel. Compression of the lumen of the true channel producing true aortic stenosis occurred in 10 patients. Re-entry tears were identified in 8 patients (44%) and were located in the aorta in 5, common iliac in 1, and common femoral artery in 1. Four patients had an atherosclerotic fusiform abdominal aortic aneurysm.

Operative Cases

Clinical and necropsy findings in the nine patients who underwent operation for aortic dissection are summarized

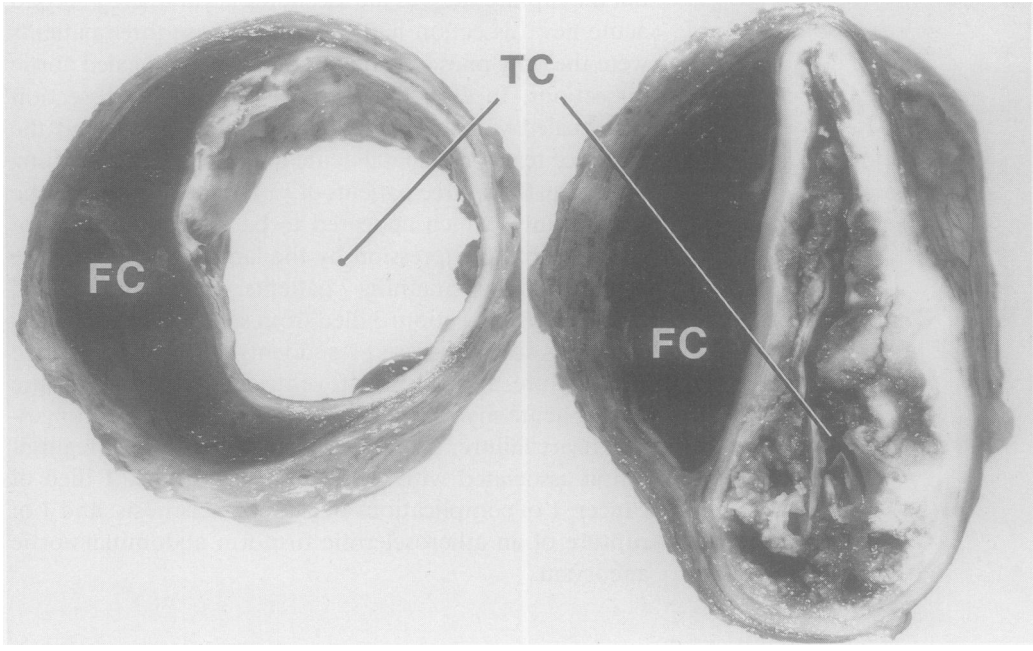
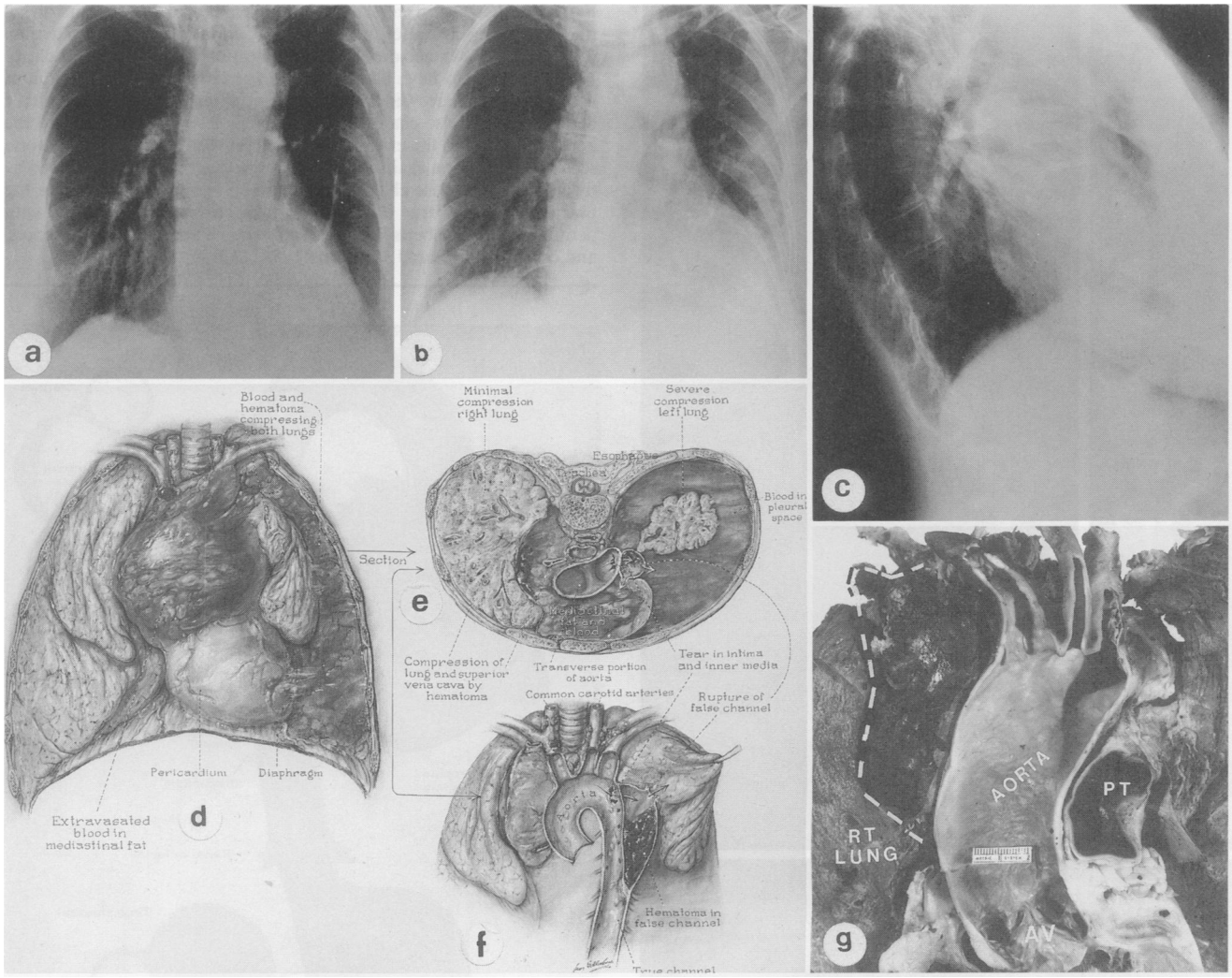


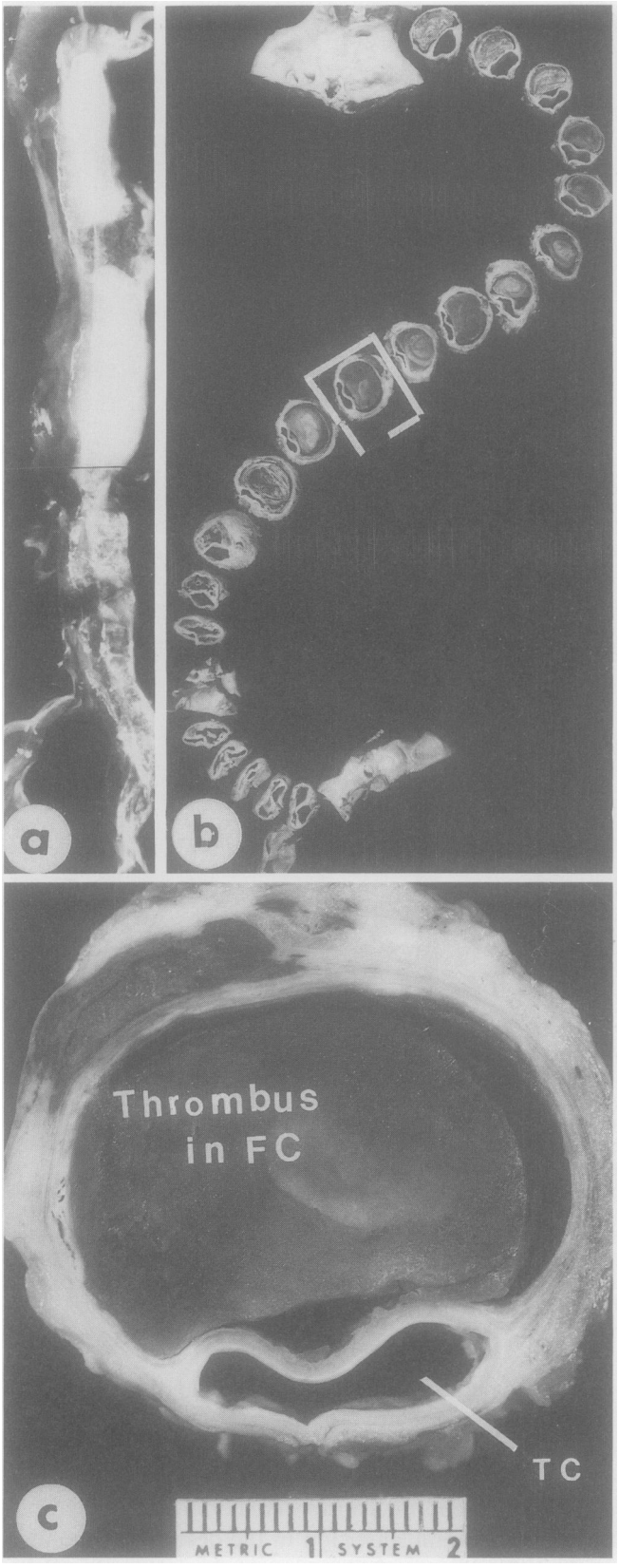
FIG. 3. (Case 11, Table 1). Photographs of two cross-sections of the descending thoracic aorta of a 71-year-old man (GT #82A-109) who died with renal failure 3 weeks after acute aortic dissection originating at the isthmus and extending to the level of the diaphragm. The false channel (FC) did not rupture. Death occurred from renal and cerebral dysfunction that resulted from pharmacologically induced hypotension. Left: The true channel (TC) is wide open but the false channel is filled with thrombus. Right: A more distal portion of aorta showing severe atherosclerosis of the true lumen and compression of its lumen by the thrombus in the false lumen.



FIGS. 4a–g. (Case 12, Table 1). Acute dissection in a 76-year-old woman (GT #77A-173). The entrance site was the descending thoracic aorta with extravasation of blood into anterior mediastinal tissues simulating a dissection from a tear in the ascending aorta. The patient had been well until 14 days before death, when she noted epigastric pain and nausea. A chest radiograph (a) showed an abnormally shaped thoracic aorta. The electrocardiogram showed left ventricular hypertrophy, and her blood pressure was 200/100 mmHg. Because of previous esophageal stricture, her chest pain was believed to be related to the esophagus. She felt well thereafter until the day of death, when she suddenly noted dyspnea and profuse sweating; repeat chest radiographs (b and c) showed further widening of the mediastinum and markedly diminished lung space in the left hemithorax. Her heart stopped shortly thereafter. At necropsy the left lung was collapsed and most of the left hemithorax was filled with blood (d and e). A localized descending thoracic aortic dissection was present (f) and the entrance site into the false channel was at the aortic isthmus. The caudal two thirds of the false channel was filled with thrombus and the outer wall of the false channel was ruptured. Before the final extravasation of blood into the left hemithorax, blood appears to have dissected more slowly into the mediastinal tissues lying to the right of and anterior to the aorta. Hemorrhage into these mediastinal tissues on chest radiographs simulated dissection of the ascending aorta. (g) Close-up view of mediastinal hematoma to the right of the ascending aorta and behind the arch arteries. PT, pulmonary trunk; AV, aortic valve. (Reproduced with permission from Roberts WC. Aortic dissection: anatomy, consequences, and causes. Am Heart J 1981; 101:195–214.)

in Table 2. The ages of death ranged from 39 to 71 years (mean, 66 years); four were men and five were women. Of the 9 patients, 8 had had systemic hypertension by history and 7 had hearts of increased weight. The indication for operation in at least 7 of the 9 patients was a major complication of aortic dissection: 3 had limb or end-organ ischemia, 2 had hemorrhage into the left hemithorax, 1 had recurrent back pain with longitudinal progression of the dissection (by repeat aortogram), and 1 had aortic regurgitation from retrograde dissection. The interval from onset of symptoms or signs compatible with aortic dissection to operation was 30 days or less in all cases.

Three different procedures were performed in the 9 patients: tubular graft insertion in the descending thoracic aorta in 7 patients, obliteration of the false channel by suture in 1 patient, and a re-entry procedure in the distal portion of the dissected aorta in 1 patient.



FIGS. 5a–c. (Case 14, Table 1). Clinically silent healed dissection involving the descending thoracic and abdominal aorta in a 59-year-old man (NNMC #A76-87) who died from complications of habitual alcoholism. He was known to have had systemic hypertension. (a) Radiograph of aorta showing a narrowed true channel and thrombus plus heavy calcific deposits in the false channel. The entrance site was just distal to the aortic isthmus and the exit was in the left femoral artery. (b) Transverse sections of the aorta again showing that the true channel is narrowed by thrombus in the false channel. (c) Close-up view of the section in the brackets in b. TC, true channel; FC, false channel. (Reproduced with permission from Roberts WC. Aortic dissection: anatomy, consequences, and causes. Am Heart J 1981; 101:195–214.)

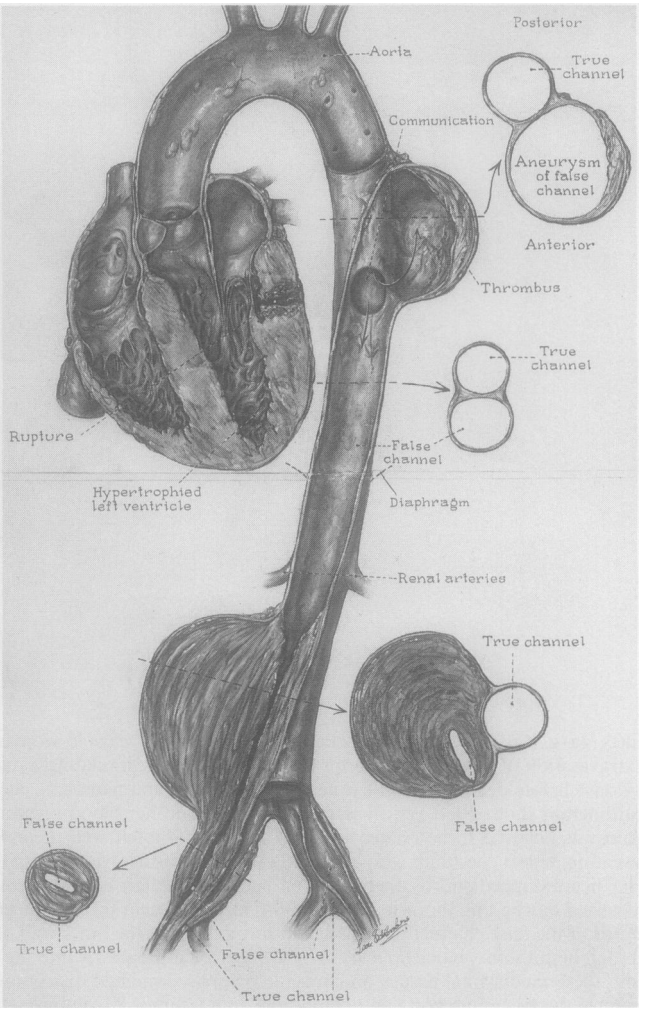
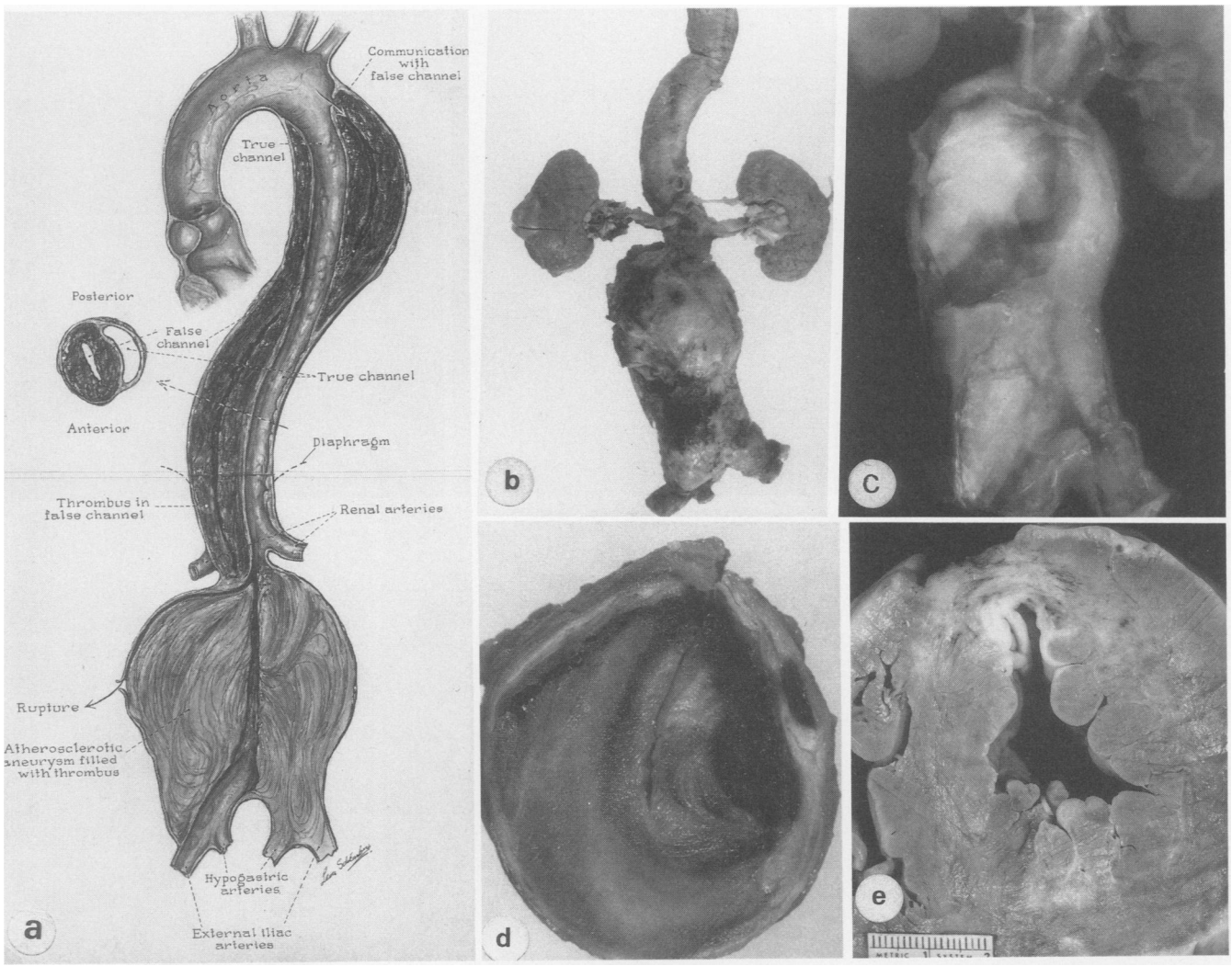


FIG. 6. (Case 17, Table 1). Asymptomatic healed dissection of the descending thoracic and abdominal aorta in a 63-year-old man (GW #8399) who died of rupture of the wall of left ventricle during acute myocardial infarction. There was no clinical evidence of an aortic dissection. He was known to have had systemic hypertension. He was asymptomatic until about 12 hours before death when he had the onset of chest pain of acute myocardial infarction. The drawing shows the entrance site into the false channel in the descending thoracic aorta and the false channel just caudal to this site developed a saccular aneurysm. The false channel in the abdominal aorta also became aneurysmal and filled with thrombus. The dissection extended into both common iliac arteries. The acute transmural myocardial infarct ruptured, causing massive extravasation of blood into the pericardial sac. (Reproduced with permission from Roberts WC. Aortic dissection: anatomy, consequences, and causes. Am Heart J 1981; 101:195–214.)

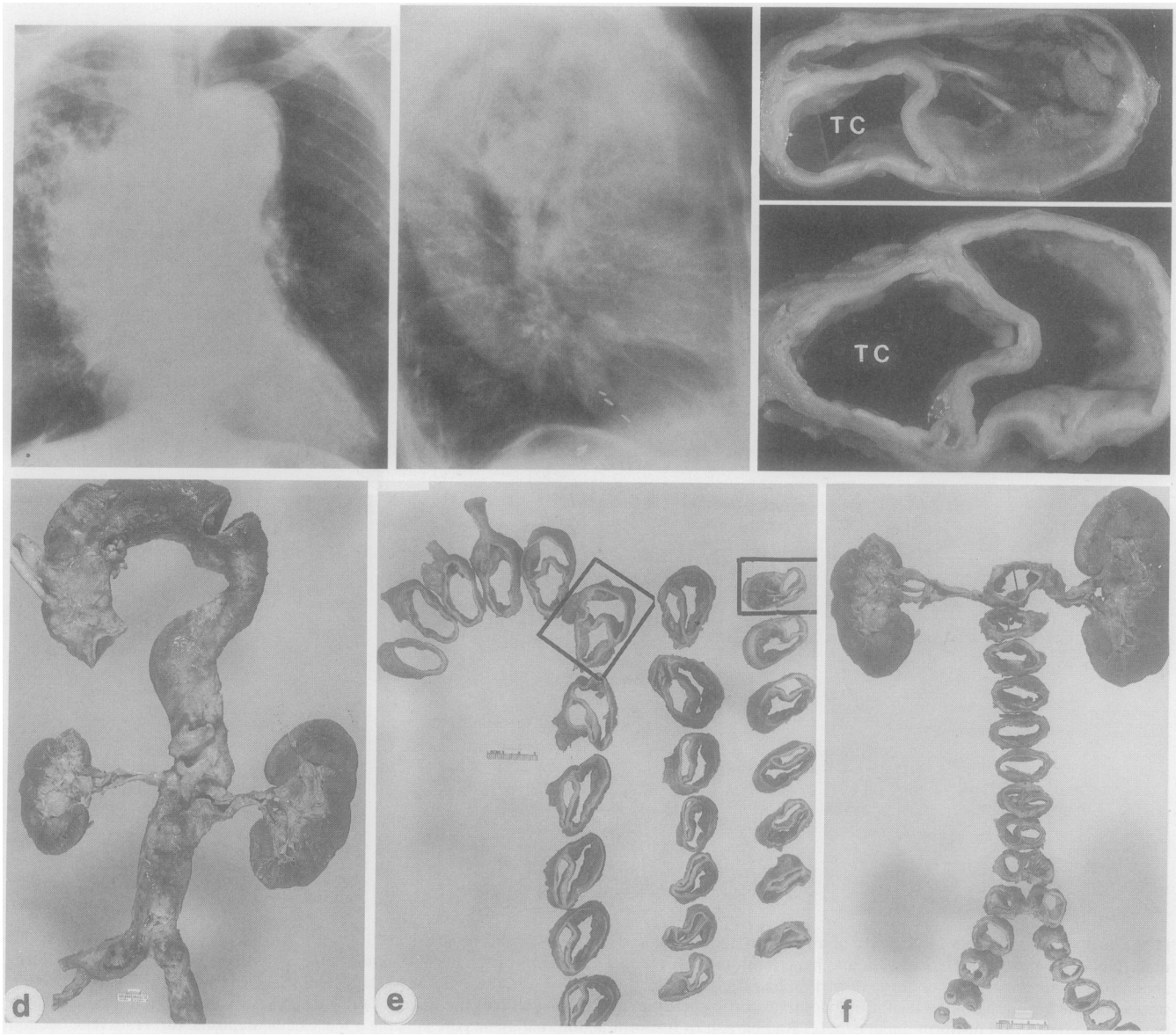


FIGS. 7a–e. (Case 21, Table 1). Rupture of a fusiform abdominal aortic aneurysm and healed dissecting aortic aneurysm together in a 67-year-old man (NNMC #A76-211). He had systemic hypertension for years and more than one acute myocardial infarct that had healed. Neither fusiform abdominal aortic aneurysm nor aortic dissection had been diagnosed clinically. (a) Drawing of the locations of the aortic aneurysms. The dissection stopped at the beginning of the fusiform abdominal aortic aneurysm and began just past the origin of the left subclavian artery. (b) Exterior view of the aorta, kidneys, and fusiform aneurysm. (c) Radiograph of fusiform abdominal aneurysm showing a few calcific deposits. (d) Transverse section of the abdominal aneurysm showing a severely narrowed lumen. (e) Transverse section of left ventricle showing a healed transmural infarct. (Reproduced with permission from Roberts WC. Aortic dissection: anatomy, consequences, and causes. Am Heart J 1981; 101:195–214.)

Five of the nine patients died within 9 days of operation: the mode of death was hemorrhage in 3, acute renal failure with anuria (both before and after operation) in 1, and unclear in 1. All 5 patients who died early had major preoperative complications of dissection that probably would have resulted in death: hemorrhage in 2 patients, organ ischemia in 2, and progression of dissection in 1. Four of the nine patients died late (at 8 to 84 months). The mode of death was aortic hemorrhage in three patients; infection in two of them led to anastomotic breakdown; and in the third patient, an unreplaced portion of false channel ruptured. The only patient in whom the aorta or aortic anastomosis did not rupture died of infection.

Discussion

Analysis of the aforementioned 40 necropsy patients with aortic dissection resulting from an entrance tear in the descending thoracic aorta disclosed that 13 of the patients died within 30 days of the event without operative intervention, 9 others underwent operative therapy during the acute phase because of complications of the dissection, and in 18 patients the dissection healed without operative



FIGS. 8a–f. (Case 23, Table 1). Clinically silent aortic dissection after resection of an abdominal aortic fusiform aneurysm in a 69-year-old man (DCVAH #78A-178) who died of bronchogenic carcinoma. The abdominal aorta was replaced with a graft about 1 year before death. Dissection of the aorta stopped at the beginning of the graft. There was no history of systemic hypertension, and the heart weighed only 330 g. Chest radiographs (a and b) shortly before death; aorta, common iliac arteries, and kidneys (d); cross-sections of aorta to the renal arteries (e) and below (f), which include the Dacron graft. The dissection began at the aortic isthmus. The false channel is larger than the true channel. Thrombus is present in some portions of the false channel (c). Close-up views of the two-bracketed cross-sections are shown in e. (Reproduced with permission from Roberts WC. Aortic dissection: anatomy, consequences, and causes. Am Heart J 1981; 101:195–214.)

intervention. The nine patients who underwent operative therapy during the acute phase probably would have died from the acute complication that led to the operation. Thus, of the 40 patients, less than one half (45%) survived the acute period to 'heal' the dissection.

Compared to dissections resulting from tears in the ascending or transverse aorta, dissection with the entrance tear in the descending thoracic aorta has the highest frequency of healing. Among our 182 necropsy patients with aortic dissection, the entrance tear was the ascending aorta in 128 patients and only 8 (6%) healed; the entrance tear was the transverse aorta in 12 patients and none healed. Other comparative information virtually is impossible to acquire from previous publications because the site of the entrance tear was not the basis for classifying the type of dissection, and the site of the entrance tear was not always

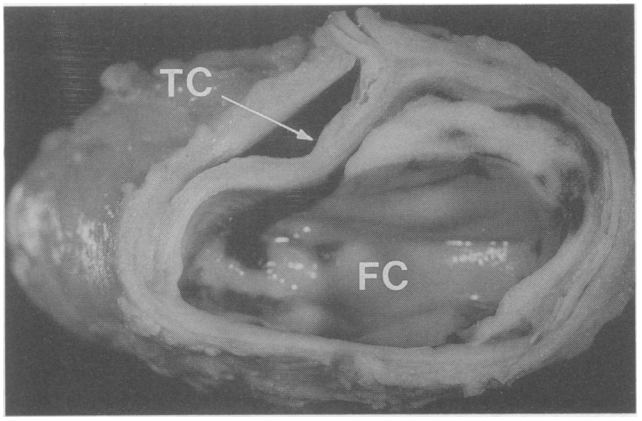
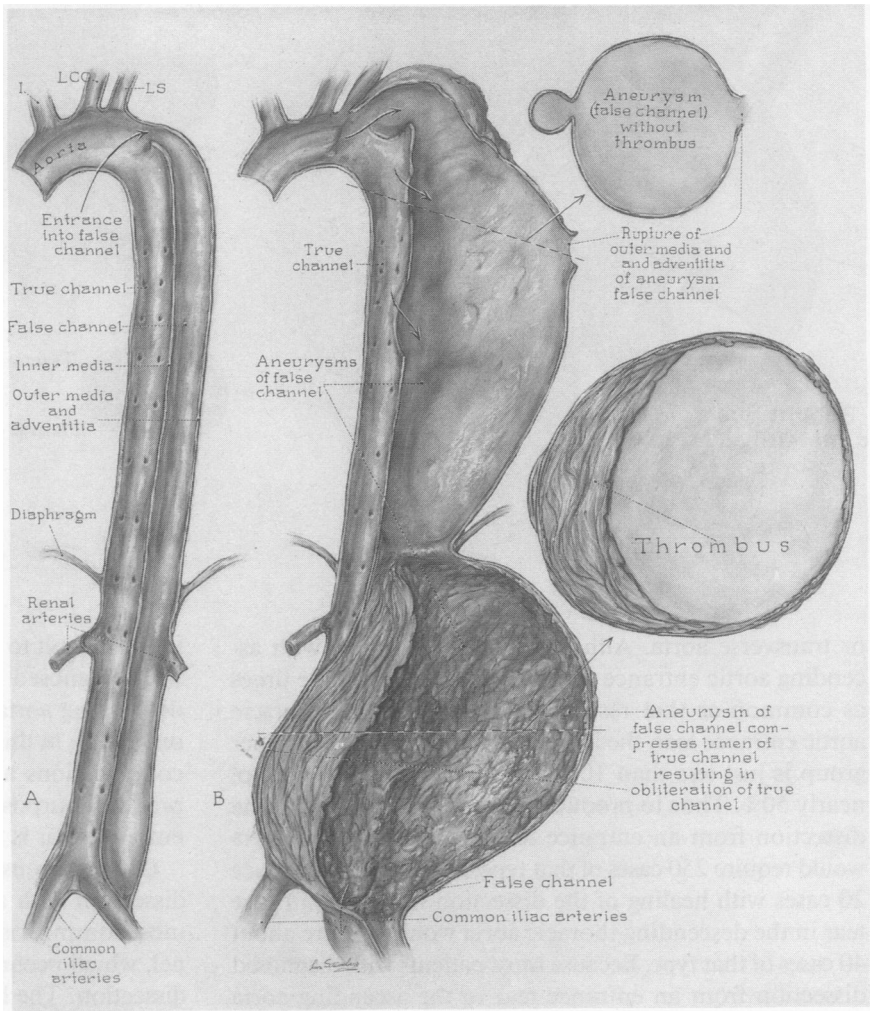


FIG. 9. (Case 24, Table 1). Cross-section of dissected descending thoracic aorta in a 76-year-old woman (GT #76A-147) who died of renal failure. The true channel is stenotic from compression from thrombus in the false channel. FC, false channel; TC, true channel.

clarified. In Shennan's 1935 monograph,[4] for example, of 309 necropsy cases of aortic dissection reviewed (only 28 of which actually were studied by the author), 233 (75%) patients died acutely, and in 79 (25%) patients the dissection healed. Of the latter 79 patients, the dissection did not involve the ascending aorta in 46 (58%), but tears involving either the arch or descending thoracic aorta were not separated. Furthermore the percentage in whom the dissection was retrograde was not delineated. Nevertheless this older report and others[5-7] indicate that dissection resulting from tears in the ascending aorta infrequently heal and that those originating from tears in the descending thoracic aorta heal more frequently.

Late complications, *i.e.*, those after 'healing' of aortic dissection, are far more common with dissection caused by an entrance tear in the descending thoracic aorta then in those caused by an entrance tear in the ascending aorta



FIGS. 10A and B. (Case 26, Table 1). Drawing of healed dissection with entrance tear at the isthmus in an 80-year-old woman (GW #7754). (A) Early after healing. (B). At time of necropsy. The false channel reached enormous size and compressed the true channel. The abdominal aortic aneurysm was of the false channel, although it might be confused easily with an atherosclerotic fusiform abdominal aortic aneurysm. A re-entry tear was located in the left common ilian arter. I, innominate artery; LCC, left common carotid; LS, left subclavian.

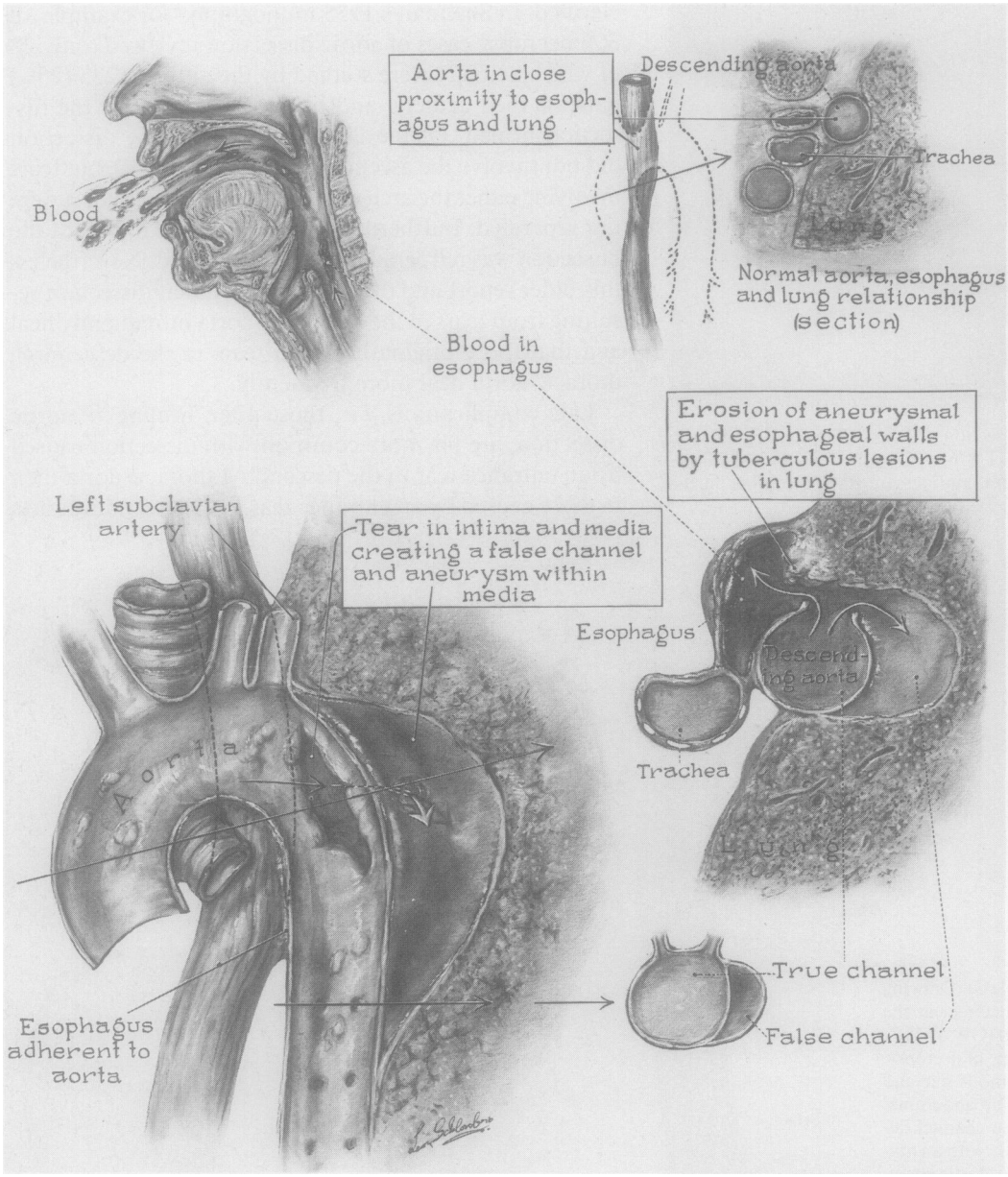


FIG. 11. (Case 28, Table 1). Drawing of a dissected descending thoracic aorta with entrance tear in descending thoracic aorta in a 91-year-old woman (GT #80A-126). She collapsed and was found with blood ejecting from her mouth. Necropsy disclosed that the lungs were infected by tuberculous bacilli and that the left lung was adherent to the descending thoracic aorta. The pulmonary infection was the medium that allowed the aorta to rupture into the esophagus, to which it was adherent. (Reproduced with permission from Roberts WC. Aortic dissection; anatomy, consequences, and causes. Am Heart J 1981; 101:195–214.)

or transverse aorta. Although aortic dissection with ascending aortic entrance tear is just more than three times as common as that resulting from descending thoracic aortic entrance tear, the frequency of healing in the former group is just less than 10% and that in the latter group, nearly 50%. Thus to produce 20 cases with healing of the dissection from an entrance tear in the ascending aorta would require 250 cases of that type; in contrast to produce 20 cases with healing of the dissection from an entrance tear in the descending thoracic aorta would require about 40 cases of that type. Because most patients with diagnosed dissection from an entrance tear in the ascending aorta now undergo immediate operative therapy, data on healing is difficult to obtain. In contrast, because most patients with diagnosed dissection from an entrance tear in the descending aorta do not undergo operative therapy, data on healing in this group is easier to acquire. Consequently complications after healing of the aortic dissection, for practical purposes, are limited to the group in which the entrance tear is the descending thoracic aorta.

Of the various late complications after healing of aortic dissection with a descending thoracic entrance tear, the most common is aneurysmal formation of the false channel, which occurred in all 18 of our patients with healed dissection. The larger the aneurysm of the false channel, the greater the likelihood of rupture of the wall of the false

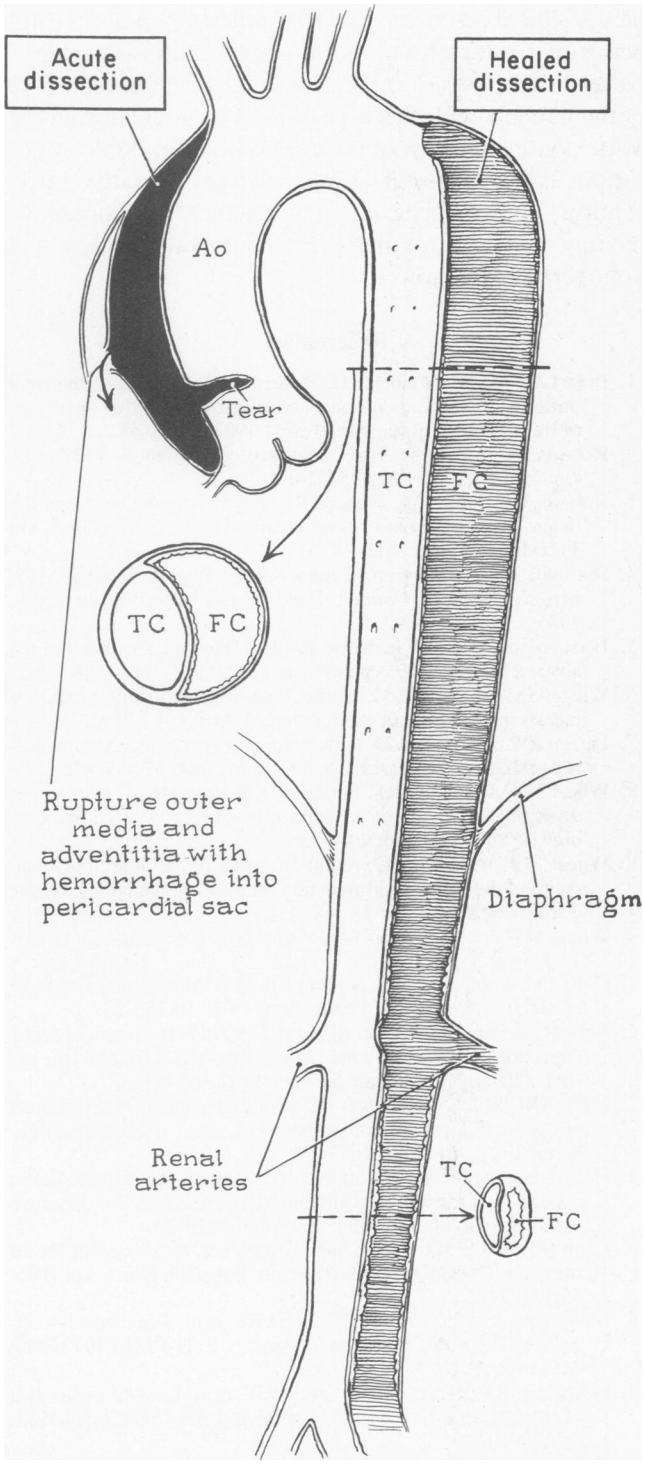


FIG. 12. (Case 29, Table 1). Drawing of aorta in a 48-year-old man (NCBH #A15-837) who died of rupture of the wall of the false channel after acute dissection with an entrance tear in ascending aorta. Found incidentally at necropsy was a healed dissection of the entire descending aorta with an entry tear at the isthmus. The patient's son died at 28 years of age, also from acute aortic dissection. (Reproduced with permission from Warnes CA, Kirkman PM, Roberts WC. Aortic dissection in more than one family member. Am J Cardiol 1985; 55:236–238.)

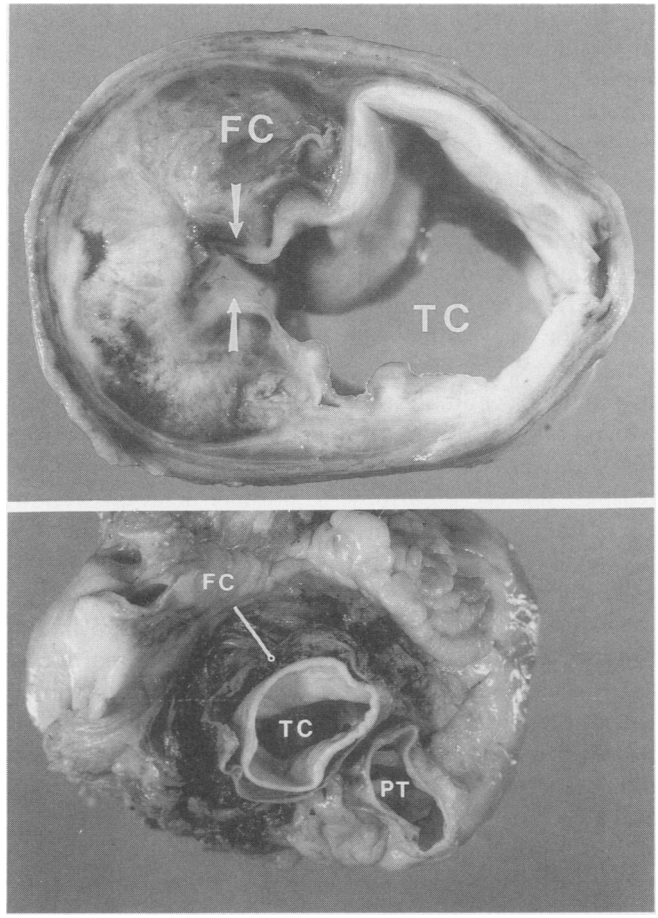


FIG. 13. (Case 31, Table 1). Photograph of aorta (upper) and heart (lower) of a 63-year-old man (SH #A86-34) with systemic hypertension who died of ruptured acute dissection with entrance tear in the ascending aorta. At necropsy, a healed dissection with an entry tear at the isthmus also was present. Upper: Cross-section of the dissected, healed descending thoracic aorta. The false lumen (FC) is filled with organized thrombus and it does not significantly compromise the true lumen (TC). The arrows designate the entrance tear. Lower: View of heart from above showing the acute dissection with false channel filled with recent thrombus.

channel, which occurred in five patients. The fact that the wall of the false channel may rupture late has received little attention previously. The presence of a re-entry tear did not appear to protect the healed dissection group from rupture of the false channel. Of the 18 patients with healed dissection, 9 had a re-entry tear: 3 ruptured the wall of the false channel exteriorly and 6 did not. Because thrombus was so frequently present (15 patients) in the false channel in the 18 patients with healed dissection, no relation was found between its presence and aneurysmal formation of the false channel or an exit tear in the partition between the true and false channels. Renal failure (3 patients) appeared to be the consequence of compression on one or both renal arteries by the aneurysmally dilated false channel. Obstruction of the lumen of the true

channel occurred in 11 of the 18 patients with healed dissection. All 11 patients with true channel stenosis had thrombi in the false channel, and none of the three patients without false channel thrombus had true channel stenosis. The narrowing of the true channel appears to be the consequence of compression from the thrombus-containing false channel. Hemodynamic confirmation of true channel stenosis in this circumstance is not available. The wall of the false channel calcified in 1 of the 18 patients. Because the acute dissection in this patient was clinically silent, the interval from the acute dissection to death is unknown. And finally an acute dissection with an entrance tear in the ascending aorta occurred in 3 of the 18 patients with a healed dissection from a descending thoracic aortic entrance tear. That healed aortic dissection may later, for reasons unclear, predispose to another acute dissection elsewhere in the aorta also has received little attention previously.

The aorta with dissection, of course, is not immune to other diseases of the aorta. Of our 40 patients, 5 had a fusiform atherosclerotic aneurysm of the abdominal aorta, and in all of them the dissection terminated at or proximal to the atherosclerotic aneurysm. One of the forty patients had typical clinical features of the Marfan syndrome. Although the ascending aorta was dilated in this patient, the entrance tear was in the descending thoracic aorta only, and the dissection involved only the descending thoracic aorta. In contrast to their occasional occurrence in patients with dissection from an entrance tear in the ascending aorta, none of the 40 patients with an entrance tear in the descending thoracic aorta had a bicuspid aortic valve or aortic isthmic coarctation.

Throughout this article we focused on the location of the entrance tear and not on the portion of the aorta involved by the dissection. We believe this focus is appropriate because the site of the entrance tear is the major determinant of the frequency of healing of the dissection, the mode of death, and the operative approach.

The relatively high frequency of healing of aortic dissection with a descending thoracic aortic entrance tear and the relatively high operative mortality rate in this group caused Wheat and associates[8] in 1965 to advocate nonoperative intervention for this type of dissection.[9,10] This viewpoint, with some exceptions,[11–15] has dominated therapy for aortic dissection of this type since that time.[16–21] Operative mortality rates have remained relatively high (compared to those for dissection originating from a tear in the ascending aorta), but usually operation was not carried out for this type of dissection unless a recognized complication occurred. Thus the mortality rate for early operative intervention for dissection with a descending thoracic aortic entrance tear without complications may be considerably lower than that when operation is not performed until one or more complications develop.[12] Nevertheless, without operative therapy, more than 50% of the patients with this type of dissection die during the acute phase and most of those who heal develop complications that in themselves later warrant operative intervention. Thus we believe that, in balance, early operative therapy will allow a much greater survival rate than will nonoperative therapy.

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