ACUTE AORTIC DISSECTION

Medical Grand Rounds

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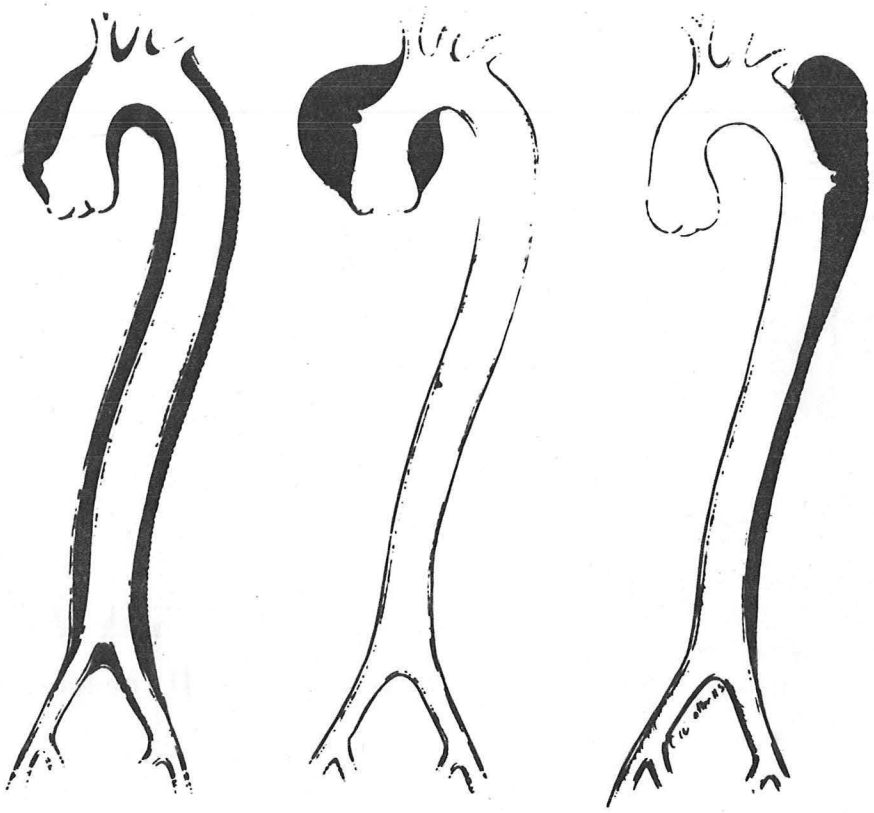

"Diseases desperate grown
By desperate appliance are relieved
Or not at all."

Shakespere
Hamlet, act IV, scene iii

Acute aortic dissection was first found by Vesalius in 1557 (1). The first description of separation of the layers of the aortic wall was by Nichols in 1755 who performed the autopsy on King George II of England (2). The first accurate description of a dissection was by Maunoir in 1802:

> "The intima ruptures at one point and the adventitia forms a pocket and it alone resists the blood effusion which progresses following dehiscence of the intima. The pocket enlarges, the blood dissects occasionally around the entire arterial circumference and the artery finds itself in the center of the aneurysm, bathing in the aneurysmal blood" (3)

During the 1800's Hodgson found that injection in the aortic wall produces dissection of the intima (4). By 1839 Pennock had produced experimental dissection in normal arteries by saline injection into the media (5). Also during the 1800's the role of hypertension, medial necrosis, and catecholeamines were described (6). In 1907 Waterman defined 3 basic causes of aortic dissection - hypertension, adrenaline, and ischemic necrosis secondary to vasa obstruction (7). Shennan, however, was one of the major contributors to our understanding of the pathophysiology of dissections (8). Shennan analyzed in detail 300 cases as far as their history, pathophysiology, pathology, prognostic features, classification, associated syndromes, and felt that dissection was secondary to a combination of many factors. The next major advance was in 1955 when DeBakey, Cooley, and Creech reported 4 successful of 6 attempts at suturing the distal intima to the outer media then reconnecting the aorta either primarily or with a graft (9). However, the overall results of dissections remained dismal until 1965 when Wheat and Palmer et al described the medical management of dissections (10). The major problem in dissections today is to know on which patients and when should surgery be performed and on which should medical management be utilized. Recently Anagnostopoulos (6) and Wheat (11) have described rational approaches to therapy for this disease and these approaches will be the major focal point of this presentation.

Incidence

The reported incidence of acute aortic dissection appears to be 5-10 cases/million/year (11). However, this is probably a gross underestimation due to the fact that most cases die unrecognized. The majority of patients who have dissection also have hypertension and/or atherosclerotic cardiovascular disease and when these patients die they are assumed to have a myocardial infarction and no post-mortem is performed. Older autopsy series reported dissection as cause of death in 1 in 714 autopsies (12,13) to 1 in 128 autopsies (14). If you assume that for every patient

dying with dissection who is autopsied there are at least 2 more and that the aortic dissection is found in one per 100 autopsies, then the rate of dissection is about 60,000/year (6). In the centers that are extremely attuned to the problem of dissection they find one acute dissection for each 10-20 acute myocardial infarctions admitted; hence the incidence of acute dissection may be 5-10% of the rate of acute myocardial infarctions. Therefore, the estimation of 60,000/year or 3/10,000 population may be a gross underestimation (6). If these rates are correct and there is suggestive evidence that they are, then approximately 375 aortic dissections/year occur in Dallas; only 55 are diagnosed. Hence more awareness of the possibility of dissection is imperative to reduce the high incidence of undiagnosed cases.

In the 50-60 age range, aortic dissections occur 2-3 times more frequently in men (8,11,12,15). Below the age of 40 there is no sexual predisposition; pregnancy accounts for ½ of the dissections in this age range (16). Women predominate in the over 60 age range (17). Dissection is more common in black than other groups possibly due to increased incidences of hypertension and Marfan's syndrome (11,15).

Histology

To better understand the pathophysiology of acute aortic dissection, a review of the basic histology of the aorta is warranted. The normal human aorta is about 25 mm in diameter and only 2 mm thick (6). The wall is considerably thinner for the diameter than are other arteries. The wall is composed of three layers, the intima, the media, and the adventitia.

The intima is normally about 130μ thick. The intima is composed of an inner layer of endothelial cells. Underneath the endothelial cells are a thin layer of fibroblasts and collagen fibrils. Beneath this thin layer are a fenestrated membrane of elastic fibers and bundles of smooth muscle. Though not as well defined, this elastic layer corresponds to the elastica interna of smaller arteries (18).

The most important layer is the media. It is the media that gives the aorta its strength, and it is this layer which is important in the pathophysiology of dissection. The media is 1.12 mm in thickness and organized in a layered fashion. Each layer of the media is composed of thin fibers of elastin and collagen and longitudinally arranged smooth muscle cells bordered by thick bands of elastic fibers. These smooth muscle cells are called medial cells and are attached to the thick elastin

lamellae by a fine network of elastin which also connects them to the collagen network. These medial cells which were once thought to be fibroblasts until electron microscopy proved that they were smooth muscle cells are responsible for the manufacturing of the elastic and collagen fibers. These medial cells have been called a "multifunctional medial mesenchymal cell" due to the multiple functions they serve (19). The cells and fibers are embedded in a matrix of acid mucopolysaccharide (6).

Smooth muscle cell protein comprises 20% of the mammalian media (20,21). The fibers of collagen and elastin comprise 60% of aorta; the elastin is most prominent in the thoracic aorta and thins as the aorta descends into the abdomen leaving a high ratio of collagen in the abdominal aorta (22). Dark bands of elastin form concentric plates of lamellae interlaced with finer elastin fibers. Collagen fibers are arranged circumferentially and are interspersed with the elastin fibers; there are no interconnections between the collagen and elastin fibers (18). These units have been defined as "lamellar units" (18). The number of these units are proportional to the aortic diameter and medial thickness. The number of lamellar units varies from species to species; in the thoracic aorta of man there are 58, each measuring 0.018 mm (18).

The medial cells get their blood supply from either the aortic lumen or the vasa vasorum. In the thoracic aorta the intima and inner one-third of the media are supplied with necessary substrates by diffusion (23). The outer two-thirds of the media are supplied by branches of the adventitial arteries called the vasa vasorum; experimental obstruction of these vessels causes ischemic necrosis of the outer two-thirds of the media (24). Comparative anatomy of various species have shown that vasa vasorum are only present if there are more than 29 lamellar units (18).

In man the thoracic aorta has 58 lamellar units of which the inner 29 are avascular and the outer 29 are fed by the vasa vasorum. The avascular zone is 0.49 mm as is the vasa vasorum zone. The abdominal aorta only has 28 lamellar units and hence is avascular. However, unlike other species each of these units in the human abdominal aorta is much thicker than usual; if the human abdominal aorta had the normal number of lamellar units for its thickness, there would be 35 and there would have to be a vasa vasorum. If the force exerted on the aortic wall were calculated per lamellar unit, the thoracic aorta would have a tension of 2095 dynes/cm/lamellar unit while the abdominal aorta would have a tension of 3189 dynes/cm/lamellar unit (18). This high tension per unit may explain the susceptibility of the human abdominal aorta to the formation of ordinary aneurysms. However, the abdominal aorta is avascular and hence cannot have ischemic necrosis so that initiation of dissection is rare.

The outer two thirds of the media is susceptible to ischemic necrosis and this may be part of the pathogenesis of dissection. The medial cells appear to absorb low density lipoprotein and appears to cause abnormal collagen proliferation increasing the need for nutrient supply in the avascular zone. Hence this proliferation may cause a low oxygen tension in the avascular zone which further accelerates deposition of lipoproteins. This low oxygen tension may cause necrosis of the inner media and may also lead to dissection (19,25).

The adventitia is composed of loose connective tissue which blends imperceptibly into the surrounding connective tissue (26).

The aorta does not reach its adult configuration until 25 years of age (26). At birth the thoracic aorta has 34 lamellar units of which 29 are avascular. By age 25 all 58 units are present; this may explain the rare incidence of dissection before the third decade of life.

Fluid Mechanics and Elastic Properties

A brief look into a few principles of fluid mechanics may help our understanding of the pathophysiology of dissection. Flow in the aorta is normally laminar in nature. Laminar flow has its greatest velocity in the center of a vessel and the slowest flow at the periphery (Figure 1).

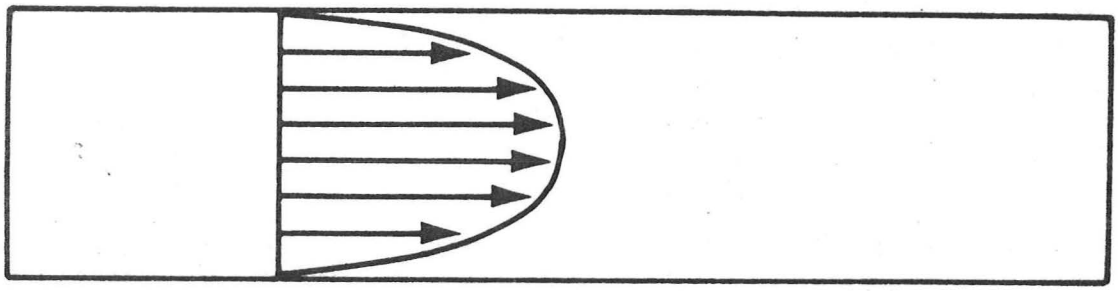

Figure 1. Laminar flow; relative velocity of particles (6)

Laminar flow is important as if the flow becomes turbulent more stress is placed on the wall of the vessel. The faster the flow the larger the vessel must be to prevent turbulence. The stress on the wall of the aorta is determined by the law of Poiseuille

which states that the pressure gradient or frictional resistance is proportional to the reciprocal of the fourth power of the radius. Hence by doubling the radius of a vessel causes a 16-fold increase in pressure, so that an abnormal increase in the size of a vessel greatly increases the stress on the vessel. Thus one factor in the pathophysiology of dissection may be dilatation of the aorta secondary to some disease process which greatly increases the stress on the vessel (6).

A second consideration is Bernoulli's law which states that at any two points along the same streamline in a nonviscous, incompressible fluid in steady flow, kinetic energy per unit volume, and the potential energy per unit volume have the same value. Since normally potential energy can be neglected this principle can be explained by the equation:

$$P_1 + \tfrac{1}{2}\rho v_1^2 = P_2 + \tfrac{1}{2}\rho v_2^2$$

where P = pressure at points 1 and 2, $\tfrac{1}{2}\rho v^2$ = kinetic energy, ρ = density of blood, and v = velocity of blood flow. In other words if the velocity increases as the vessel narrows then the pressure must fall (Figure 2).

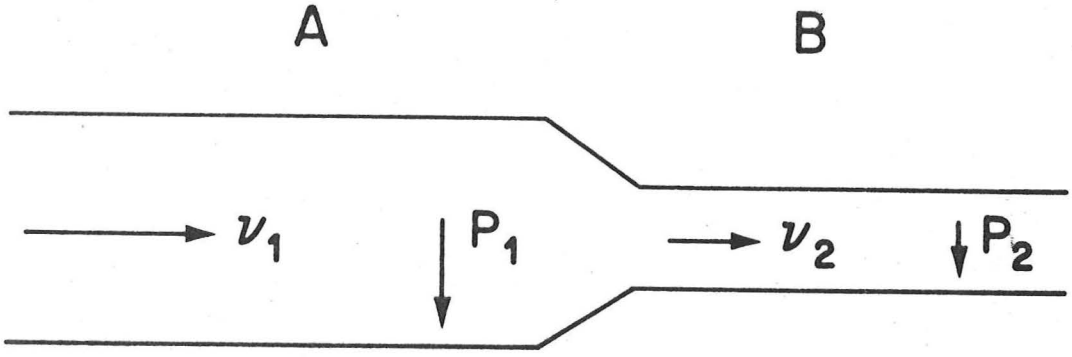

Figure 2. The application of Bernoulli's law to fluid flow. Since the sum of pressure and velocity remains constant, in section B as the velocity increases, the pressure must decrease (6).

Hence when a vessel is narrow for any reason with the same flow, velocity increases and pressure falls. This decreased pressure may produce "suctioning effect." This suctioning effect may be

important in the pathogenesis of both dissection and the formation of an atheroscleotic plaque (27). This suctioning effect is greatest at branching points of the arteries and where plaques narrow the vessel thus contributing both to the initiation and subsequent growth of the plaque.

A fourth consideration of fluid mechanics has to do with the aortic arch which is curving. The curve causes centripetal force which must be balanced by opposing forces according to Newton's third law of action-reaction. The relationship of the centripetal force is:

$$dP/dr = \rho v^2/r$$

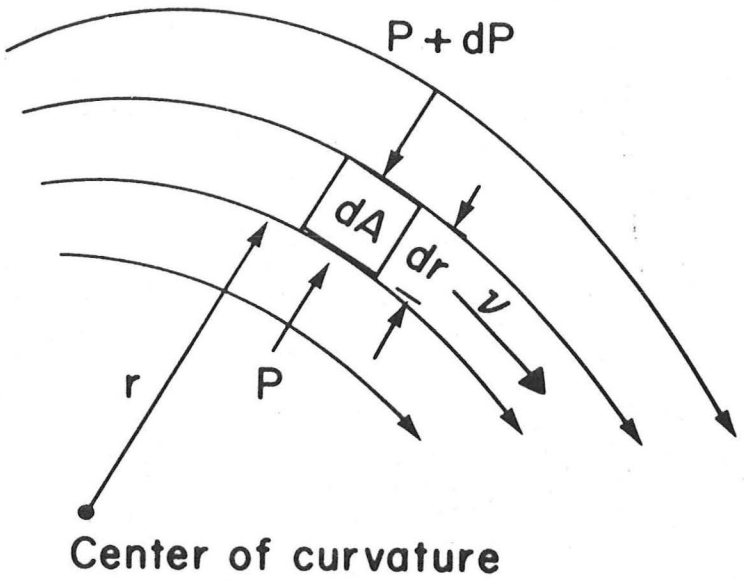

Figure 3. An element of fluid moving with velocity along a streamline of curved flow as in the aortic arch. dA = area of element of blood, dr = height of element, r = radius of curvature, P = pressure, dP = increase in pressure as radius increases (6).

This principle states that as the radius increases, velocity decreases and pressure increases. Hence the velocity is greatest and the pressure lowest at the bottom of the arch and the velocity slowest and pressure greatest at the top of the arch as shown in Figure 4:

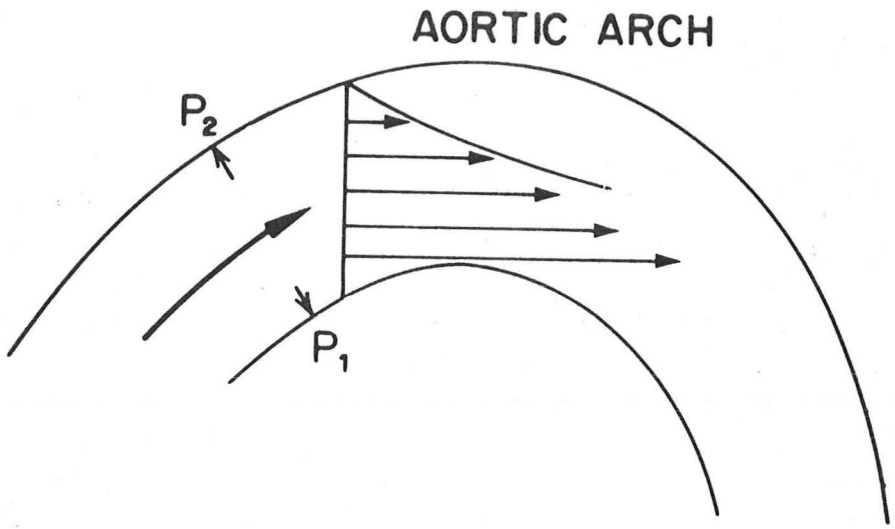

Figure 4. The effect of an increase in radius of curvature is 1) to decrease velocity and 2) to increase lateral pressure (6).

All of these factors are important considerations in considering the stress on the aortic wall. By using these principles there are two major stress points in the aorta. One point is in the ascending aorta on the lateral wall above the aortic valve and the other is on the lateral wall of the descending aorta just distal to the takeoff of the left subclavian artery.

Another physical factor of importance is the elastic properties of the aorta. A perfectly elastic substance will stretch linearly with increasing force. The aorta has a sigmoid response to stress (Figure 5). At low levels of force the aorta is very distensible. At moderate levels of force the aorta is less distensible and at high levels of force the aorta is very stiff. This is due to the influence of the elastin fibers and smooth muscle cells at low and moderate forces but limitation at high force by the collagen fibers which are much less elastic. This distensibility at low pressures allows the thoracic aorta to change in diameter by 6%. The abdominal aorta which has more collagen to elastin can only expand by 2%. This varying stress-strain relationship tends to limit expansion of the aorta below levels where high wall stress might occur according to the law of Laplace (28-33).

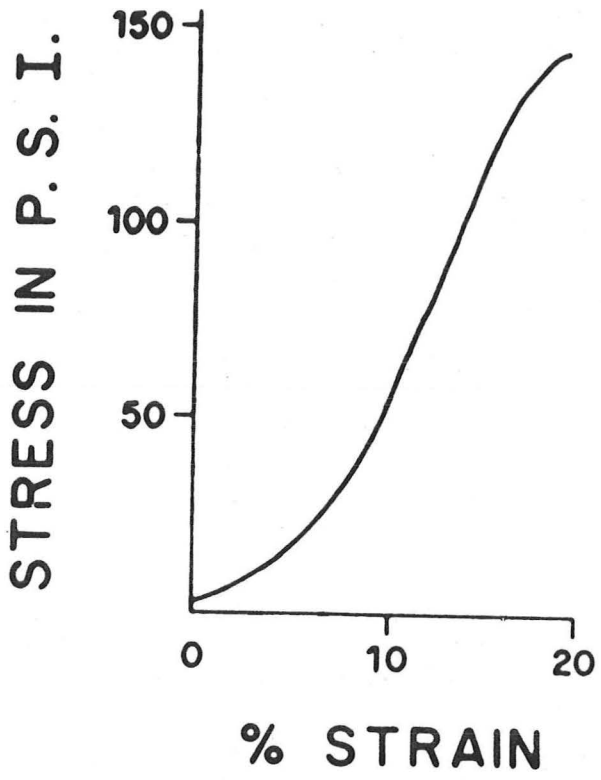

Figure 5. The stress-strain behavior of the human aorta. The sigmoid shape indicates that the aortic wall resists stretching the more it is stressed; i.e., it becomes stiffer, (P.S.I. = lbs/in^2). (6).

Pathology

Aortic dissection usually occurs in patients whose aorta have extensive medial disease. While half of all autopsy specimens show some evidence of medial disease, only the more severe have dissection. The intimal tear generally occurs in the region of the worst medial degeneration.

In patients with dissection the aorta shows loss of elasticity, unusual friability of the aortic wall, and wrinkling of the media in areas overlying the medial degeneration (34). The intimal tear can occur at any site but is usually located in the ascending aorta (35). Over 90% of the intimal tears occur in the ascending or arch of the thoracic aorta (36,37). A sample distribution of the sites of the intimal tear were illustrated by Hume & Porter:

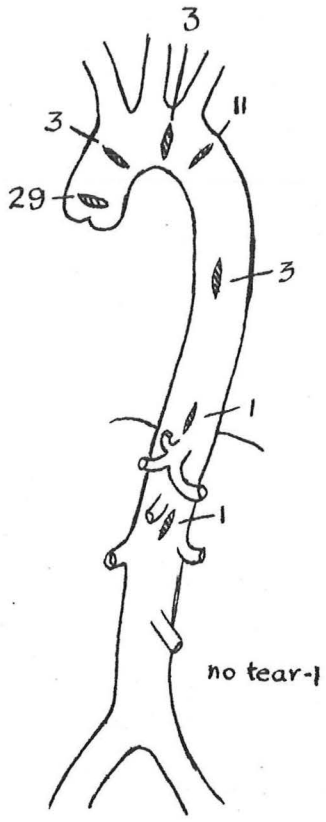

Figure 6. The location of the intimal tear in 52 cases in which this could be definitely established (38).

Several typing systems have been utilized, however the two most useful are the anatomical typing of DeBakey and the therapeutic typing of Anagnostopoulos (6,9). The DeBakey typing system consists of three types:

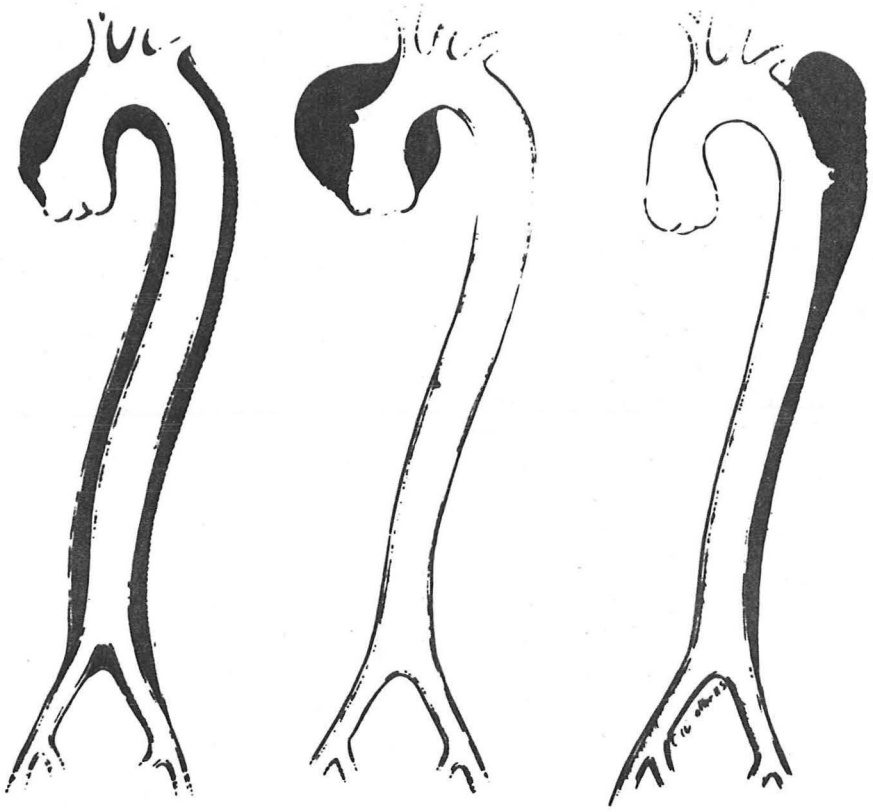

Figure 7. The classification system of DeBakey. Types I, II, and III, respectively (6).

I) Type I begins in the ascending aorta and extends at least into the arch and possibly through the descending aorta and abdominal aorta.
2) Type II begins in the ascending aorta but ends prior to the innominate artery.
3) Type III begins in the descending aorta and extends distally. Occasionally a Type III dissection may dissect retrograde through the arch and may even dissect retrograde into the pericardium.

A common finding in the predissection aorta of idiopathic cystic medionecrosis is dilatation of the aorta in areas of degeneration (39). Frequently when dilatation occurs there is aortic insufficiency due to dilatation of the aortic ring. This dilatation

increases the force on the aortic wall due to the Laplace effect which accentuates the genesis of dissection.

The dissection generally starts in the inner half of the media though on occasion it may occur in the outer half or rarely dissect between the intima and media (6,27). The dissection may extend as far as the popliteal artery (40).

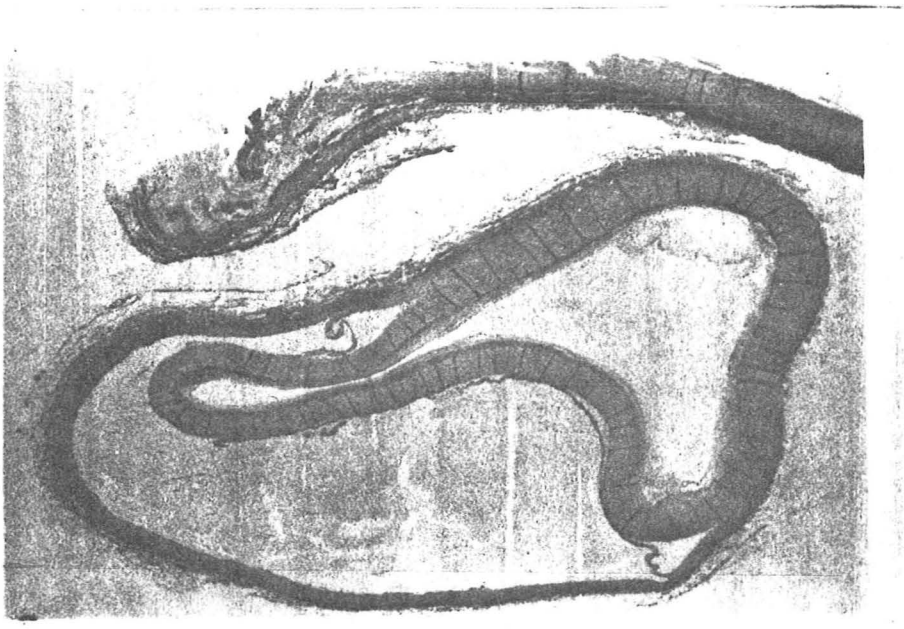

Figure 8. Dissection in the outer third of the aortic wall, note the eccentric location (6).

Though the dissection is frequently believed to be circumferential this is not usually the case. A classification of aortic abnormalities is shown here:

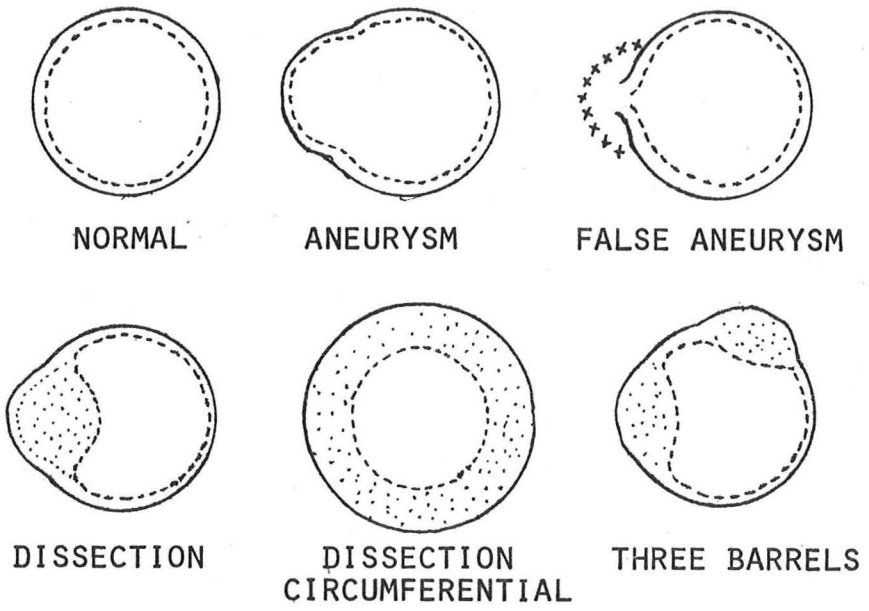

Figure 9. Schematic depiction of aortic aneurysm, and dissection. Aorta viewed in cross section. Solid line represents adventitia, broken line intima and dotted area false channel (41).

Most of the dissections are eccentric and form a double barreled aorta. Generally the dissection starts laterally in the ascending aorta, tends to go just posterior to the head and arm vessels, then goes down the lateral surface of the descending aorta. As the dissection passes the diaphragm it tends to swing posterior. Though much less common dissections are found on the medial aspect of the aorta (41).

The dissection may involve any branch of the aorta. The methods of involvement are shown below:

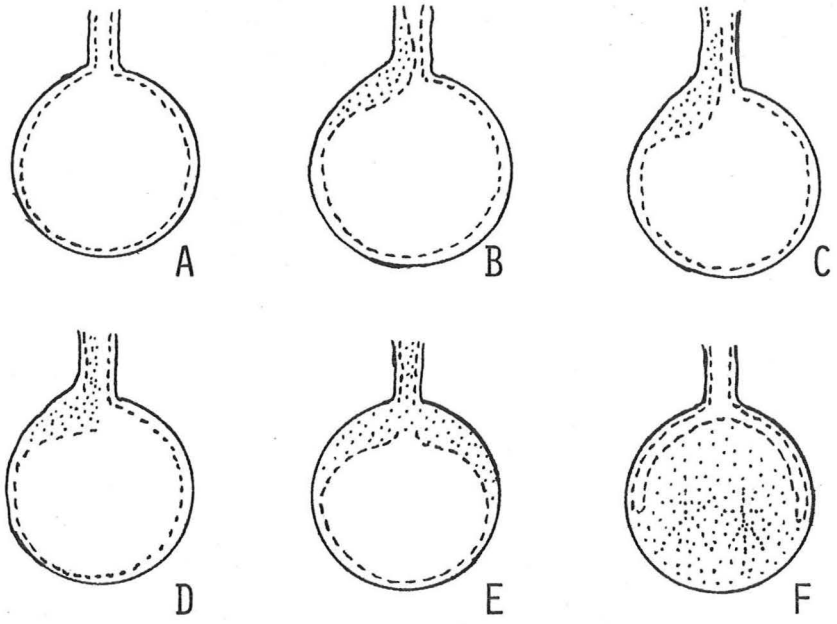

Figure 10. Diagrams of various types of branch involvement. Aorta viewed in cross section at the orifice of an aortic branch. (A) Normal. (B) Dissection extends into a branch. True lumen may be compressed or occluded by false lumen. (C) Extension of dissection into a branch with distal re-entry. (D) Intimal tear at the orifice of a branch. (E) Complete detachment of the intima at the orifice of a branch. The branch artery receives blood exclusively from false lumen, unless the intima seals off and occludes the branch. (F) No direct involvement of a branch, but the false lumen on the opposite side is approximating the orifice of a branch (41).

Coronary arteries are involved in less than 10% of the cases (37). The intercostals are the most frequently involved vessels.

The relative incidence of vessels involved are shown below:

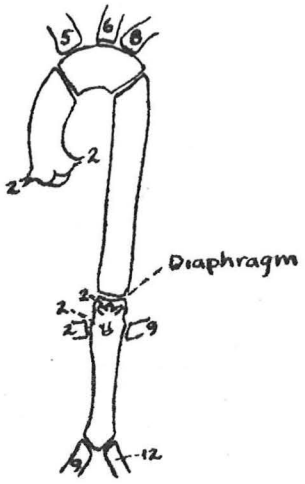

Figure 11. Vessels involved by dissecting process (37,42).

Involvement of the renal arteries may occur in as many as 20% of dissections (37%). The mechanism of renal involvement is shown below:

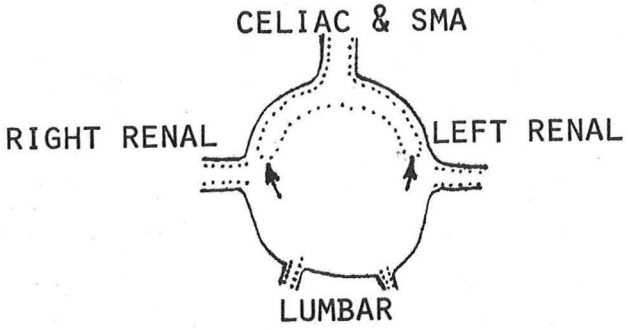

Figure 12. Schematic explanation of Figure 11. True lumen supplies celiac and superior mesenteric arteries. False lumen supplies renal arteries. Communication between true lumen and false lumen may be through the detached intima at the orifices of renal arteries (arrows) (41).

Re-entry of the aortic dissection occurs in 25-45% of the cases (35,37). The most common site of re-entry is the iliac vessels. The second most common site of re-entry is the neck vessels.

The microscopic appearance of the aorta reveals that the lesion starts in the inner half of the media but may start in the outer one-half to one-third. Simple muscle degeneration occurs first; the cells become round and detach from the elastic fibers. Adenosine triphosphatase activity is lost but glucose-6-phosphate dehydrogenase and lactate dehydrogenase activity increase. These changes suggest a shift towards anaerobic metabolism and ischemia. There are no inflammatory cells. Chondroitin sulfate C accumulates in pools of mucoid material to give the complete picture of Erdheim's cystic medial necrosis. As the medial cells die, the cystic accumulations increase resulting in a collagen-poor scar tissue which greatly thins and weakens the aorta. It should be noted that these changes are present in many age matched controls but are more severe in patients with dissection (6).

The cause of death was rupture in 95% of the deaths (8). Of the ruptures 81% were intrapericardial. Rarer causes of death include heart failure (due to aortic insufficiency), asphyxia from compression of air passages, bronchopneumonia, compression of the right pulmonary artery, superior vena cava obstruction, coagulopathy, and unknown causes (6,8,43-47).

Pathophysiology

The aorta can be viewed as a tube or pipeline. To research the role of various stresses, a Tygon tube has been lined with rubber cement and then exposed to pulsatile flow (48). With this model several methods of pipeline failure have been described; these include:

1) Disrupted maintenance of the tubing.
2) Outflow obstruction.
3) Pump hyperfunction (causing increased pressure and inner wall friction).
4) Too much volume entering a small tube (hypervolemia).
5) Destructive external interference.
6) Poor quality materials or construction.
7) Direct corrosion (6).

Correlaries of each of these types of factors have been found in medial necrosis.

TABLE I

Clinical Correlation of Causes of Medial Necrosis

1. Interference with maintainance
   Experimental vasal obstruction, ? atherosclerosis, cholesterol
2. Outflow obstruction
   Hypertension, coarctation, epinephrine, endocrine
3. Hyperfunction of pump
   Normotensive "hyperkinetic", epinephrine without hypertension
4. Hypervolemia
   Pregnancy
5. Destructive interference
   Trauma, iatrogenic

6a. Poor quality materials
   Marfan's, Erdheim's, congenital cardiac anomalies, natural dissections in turkeys

6b. Poor assembly
   Lathyrism, aminonitride

7. Corrosion from within
   X-factor (unknown) or lathyrism, aminonitride, copper deficiency

These seven groups of factors are the underlying causes of medial necrosis.

Mechanical factors also have an important role in completing the dissection. Table 2 lists mechanical factors leading to dissection:

TABLE 2
Mechanical Factors Completing Dissection
(Leading to Intimal Breaking Point)(6)

1. Pulsatile flow and rate
2. Systolic thinness
3. Suction on intima
4. Shearing and friction
5. Turbulence and viscosity
6. Deforming buckling

A model for the pathogenesis might be as follows:

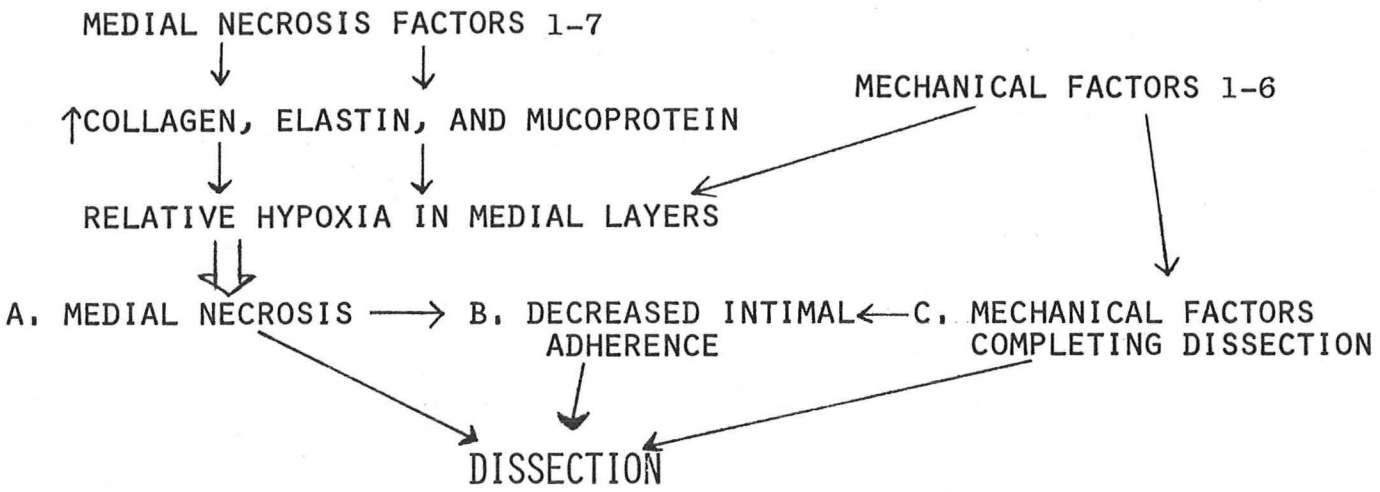

Figure 13. The pathogenesis of dissection.

As mentioned previously, acute aortic dissection occurs in a well defined area of the aortic media (24,36,48,49). The media contains necrosis, hemorrhage, or degenerative changes in the area of dissection (8,34,38,50).

Due to the large amplitude pressure waves, the human aorta is very susceptible to dissection. The pulsatile flow alternately presses out and thins the aortic wall and then "sucks" the walls inward. The compression of the aortic wall is accentuated by the stiffness of the circumferential collagen fibers (51,52). These collagen fibers limit the distensibility of the ascending aorta to about 12% and the descending aorta and arch to less (22,53,54). During systole due to the elasticity of the aorta there is clamping of the sudden compression wave of ventricular ejection (55,56). The elastin allows expansion of the aorta like a rubber balloon. The smooth muscle acts as a shock absorber (57). The collagen fibers give the aorta tensile strength (22,53,58). As systole occurs the aorta becomes radially compressed or thinned and elongated circumferentially. The same structural elements give a relative stiffness to the aorta which resists collapse and may cause a low lateral pressure resulting in a possible suction effect (51,52,57,59). Zones of increased velocity also reduce lateral pressure. Due to the fact that the aortic wall responds more slowly to luminal pressure changes, the medial layers have a net negative effect as the aortic pressure falls rapidly during diastole causing the hypothesized suction effect; there is some evidence that there are special ultrastructural elements to counteract these forces (60,61).

An additional stress on the intima is longitudinal shear. Blood flowing turbulently through the aorta tends to place a shearing force in the direction of flow due to friction between blood cells and the intima. This shearing effect is minimized by the desmosomes of the intimal cells (60,61). The points of highest shear stress are also the areas most effected by atherosclerosis (62).

The object of medical management of dissection is to reduce the mechanical factors that tned to complete the dissection. The object of surgery is to resect the worst areas of medial necrosis and provide stronger intimal adherence by suturing through the aorta from intima to adventitia.

Etiology (both clinical and experimental)

1) Embolic obstruction of the vasa vasorum.

Most dissections occur in the inner one-third to one-half of the media (8,34,36-38,50,63,64). When experimental vasal blockage either by coagulation or embolism is performed, the medial necrosis occurs in the avascular zone near the vasa vasorum, the very area where most dissections occur (24,65,66) suggesting that, even though it is in the avascular area, medial necrosis may be due to vasal obstruction.

2) Congenital cardiovascular anomalies.

Several lesions have a reported increased incidence of dissection without hypertension including PDA, ASD, tricuspid anomalies, and aortic hyperplasia (8). Aortic stenosis, tricuspid valve hypoplasia, bicuspid aortic valve, and Marfan's syndrome are associated with aortic dilatation and dissection (34,66-71). Atrioventricular valve defects have also been reported. Coarctation is a relatively common congenital cause of dissection resulting in death in 19% of all untreated coarctation patients (42).

3) Hypertension.

Hypertension or a history of hypertension has been found in 80-94% of patients with dissection (34,38,50,64,66,68).

4) Epinephrine.

Erb has reported dissection in rats given epinephrine (72). Dissections have been reported in pheochromocytoma (74,75).

5) Endocrine disease.

Dissections have been reported in Cushing's disease and pheochromocytoma (73,75).

6) Pregnancy.

Pregnancy with its hypervolemia and altered endocrine state has a very high incidence of dissection (76). Rupture occurs during the third trimester and during labor. Type I dissections are more common (75%) with Type III being the second most common site (77).

7) Secondary Obstruction of the Vasa Vasorum.

The vasa vasorum runs in all directions in the outer 29 lamellar layers (18,42-45). As the aorta expands with systole it thins and squeezes the vasa vasorum decreasing flow in them. If a patient is hypertensive then the flow occurs for a shorter period of time; if exertion or stress causes further elevation of pressure, ischemia may occur in the avascular borderline zone. Since the vaso vasorum originates from arterial branching points and not the lumen of the aorta these vessels are more susceptible to stress and secondary blockage (18,78-83).

Other "risk" factors can be additive to this stress. Obviously epinephrine and vasoactive amines accelerate the process. Hypertension obviously greatly accentuates this process. Hypertension because of its increased tension in the aortic wall and increased metabolism in the media has increased oxygen demand but a reduced delivery. The areas of highest stress are also the areas of increased atherosclerosis (18,48,51,52,56,79,80,85-91). Pregnancy has high progesterone levels which cause the medial cells to greatly increase production of mycopolysaccharides which increase wall thickness and decrease the nutrient supply to demand ratio; hence in combination with stresses of hypervolemia and possibly hypertension cause in effect restriction of vasa vasorum supply (76,85,86,92).

8) Hyperfunction of the Pump and Intimal Breakage.

Hyperfunction may contribute to dissection by increasing the level of hypertension and increasing the tear processes. One school of thought suggests that dissection is due to a vasa vasorum hematoma which ruptures into the lumen; however most dissections are in the avascular zone away from the vasa vasorum and the capillary pressures are low making it unlikely for the hematoma to propagate and rupture (8,34,38,50,68,93).

The other hypothesis to explain the intimal tear is similar to the deceleration rupture injury of the ascending aorta (93). As the aorta is fixed by the innominate and carotid arteries and the heart is free, the asending aorta undergoes flexion stresses with each heart beat which is accentuated by not being able to swing anterior-posterior due to the thorax. The high velocity pressure pulse in the aorta and the "suction effect" (53) also cause high intimal stress. Shear stresses however are probably the most important. After medial necrosis has occurred the frictional

forces cause a buckling inward of the intima; this buckling causes increased shear stress thus establishing a viscious cycle. Turbulence accentuates the process and may cause the intimal break (62). Once the tear is initiated it is then continued by the pulsatile high velocity shear forces of the aortic pulse (93-95). The pulsatile nature is important in that once a tear is formed a pulsatile pressure of 250/70 mm Hg will propagate the tear but a non-pulsatile pressure of 175 mm Hg will not. (Figure 14).

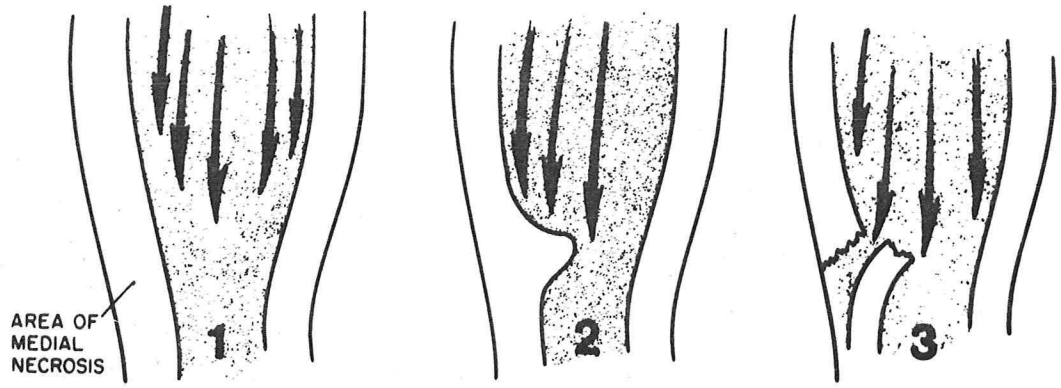

Figure 14. Tear Induction: 1, tendency of weakened area to buckle luminally with oncoming pressure wave; 2, tendency for buckled wall to be subjected to greater shearing force; 3, tear induced.

9) Hypervolemia.

Though some pregnant women with dissection have hypertension, most do not (42). Pregnancy with coarctation of the aorta has a 7% dissection and rupture rate (97).

10) Destructive interference.

Deceleration or crushing chest wall injuries have been a cause of dissection (96). Various arteriographic proceedures have been reported to be complicated by iatrogenic dissections (98-104). Open heart surgery with cannulation of the aorta or other vessel as well as arteriotomy have also been associated with dissection (17,84,105-108).

11) Poor Construction or Material Quality: Marfan's Disease Natural Dissections in Turkeys, Large Cell Aortitis.

The best known example of poor material quality is Marfan's syndrome with its associated cystic medial necrosis. These patients have a very weak media and in young normotensive patients have ascending aortic dissections which usually involve the valve ring (109-117). In Erdheim's necrosis, repair by fibrosis without vascular supply is seen; marked dilatation of the ascending aorta

with aortic insufficiency and dissection (118-123). Turkeys have naturally occurring cystic medial necrosis and have been used as an animal model for dissection (126-128). Giant cell arteritis is also frequently complicated by dissection due to medial degeneration (124,125). Syphilitic aortitis has also been associated with dissection (68).

12) Corrosion from within.

Legume intoxication is rats (lathyrism) was shown to cause dissection (129-131). Aminoproprionononitriles have been reported to cause epiphyseal plate lesions, degenerative artheritis, and dissection in rats by causing dissolution of collagen fibers (132-133). Copper deficiency in growing animals produced medial necrosis (39).

Clinical Findings

TABLE 3
Symptoms of Acute Dissection (6)

1. Pain
   - Chest
   - Submental
   - Substernal
   - Facial
   - Epigastric
   - Interscapular
   - Neck
   - Midback
   - Sacral
   - Extremities

2. Syncope
3. Coma, confusion, headache
4. Blindness
5. Dyspnea, orthopnea
6. Hemoptysis
7. Nausea and vomiting
8. Melena, hematemesis, tenesmus
9. Oliguria, hematuria, anuria
10. Hoarseness
11. Paralysis

Pain is the most common symptom. It is characterized as a "tearing", "throbbing", "lacerating", "as if something inside me tore loose", "in synchrony with each beat", "ripping", "excruciating", "burning", or "cramplike" but usually not "oppressive" (38,121,134, 135). The onset of the pain is sudden and unremitting though a few patients are pain free (6,135). The localization of the pain varies and usually reflects the area of the aorta involved, ie; pleuritic-descending aorta, flank-abdominal aorta, groin-iliac, etc. Radiation of the pain to the extremities is unusual (10-20%) probably due to "autosympathectomy" (38,121).

Neurological symptoms have been reported including paralysis and syncope occurring in 10-35% (134-136). Coma and headache were found in 10-26% (38). Transient blindness occurred in 5% (38).

Cardiorespiratory symptoms include dyspnea, orthopnea, hemoptysis, and pulmonary edema. Tracheal compression, hemothorax, and cardiac failure due to aortic insufficiency (21%) may cause dyspnea. Cardiac tamponade accounts for 81% of the mortality (38,68).

Gastrointestinal symptoms include epigastric pain, nausea, vomiting, melena, hematemesis, and tenesmus. Reversible nausea and vomiting are found in 20-33% of the patients (38).

Oliguria, hematuria, and anuria may occur with dissection due to renal artery involvement (see Figure 12, page 15)(38).

Other symptoms include hoarseness from stretch of the recurrent laryngeal nerve and Horner's syndrome due to involvement of the stellate ganglion (42).

TABLE 4
Physical Findings in Acute Dissection (6)

1. Hypertension
2. "Shocky"-cold & clammy
3. Tachycardia
4. Bradycardia
5. Rales
6. Unequal pulses
7. Absent pulse
8. Dry, warm, pulseless extremities
9. Duplication of pulses
10. Reappearing pulses
11. Tamponade
12. Unilateral or bilateral jugular venous distention
13. Cardiomegaly
14. Left hemothorax
15. Pericardial friction rub
16. Aortic insufficiency
17. Hemiplegia, paraplegia
18. Facial paralysis
19. Horner's syndrome
20. Ileus

Patients with acute dissection usually appear to be in obvious discomfort. Hypertension is found in 52-78% with a history of hypertension in 89-94% (8,38). Right bundle branch block and complete heart block have been reported secondary to dissection back into the interatrial and ventricular septum (121,237). Bradycardias of 40-50 beats/minute are seen in 10% of patients (121). Vasodilatation due to the "autosympathectomy" effect may cause a dry warm extremity with a cold, sweaty trunk (136,138). Duplication of pulses probably due to different flow rates in the true and false channel have been described (139). Examination of the neck may reveal either unilateral or bilateral venous distention; the bilateral distention may be due to either vein obstruction or cardiac tamponade (140). A pulsating neck mass may also be seen (135).

Cardiomegaly is found in two-thirds of the patients possibly due to chronic hypertension (135). Left bloody pleural effusions

lead to friction rubs. Pericardial rubs and tamponade occur in 10-34% of patients, and tamponade is the most frequent cause of death (8,135). The murmur of aortic insufficiency is found in 23-28% of patients due to either stretching of the valve ring, loss of the supporting structures for a cusp, or perforation of the dissection into the left ventricle (68,125,141,142).

The most common abdominal finding is ileus. A pulsating mass occurs in less than 3% of the patients (68,121). Temporary or permanent neurological syndromes may be seen due to intermittant obstruction of vessels by intimal flaps (42,135). Ischemic loss of reflexes due to necrosis of peripheral nerves in extremities has been found (121,135).

Laboratory Findings

In subacute and chronic dissections a normochromic, nomocytic anemia is often present (12, 68). Marked leucocytosis is usually present with an occasional leukemoid reaction seen; fever is also present at times. Fibrinolysis and a consumption coagulopathy have been described (47,143,144). Elevations of BUN and creatinine are common usually due to hypoperfusion of the renal arteries or nephrosclerosis (12). Hyperbilirubinemia has occasionally been found (145). Elevated amylase may suggest superior mesenteric artery involvement (12,38).

Electrocardiography

Left ventricular hypertrophy due to hypertension has been reported in 62% of patients. Changes of ischemia or infarction have occurred in 10-30% of cases (12,38,68,146,147). Changes of pericarditis are seen in 10% of the cases (12).

Echocardiography

Millward (148) and Nanda (149) have reported finding four simultaneously pulsating echoes due to the double lumen structure of dissection. Moothart (150) found that 5 of 6 patients with Type I dissection had this finding. Brown (151) reported three findings in dissection: 1) a widened posterior and/or anterior aortic wall, 2) parallel motion of the separated margins of the aortic wall, and 3) aortic root dilatation of 42 mm or more at end systole. However, Brown also found the same 3 findings in 5 of 10 patients with dilatation but not dissection of the aorta. Several other mimics of dissection have been reported (152). The

classical echo for dissection is shown below:

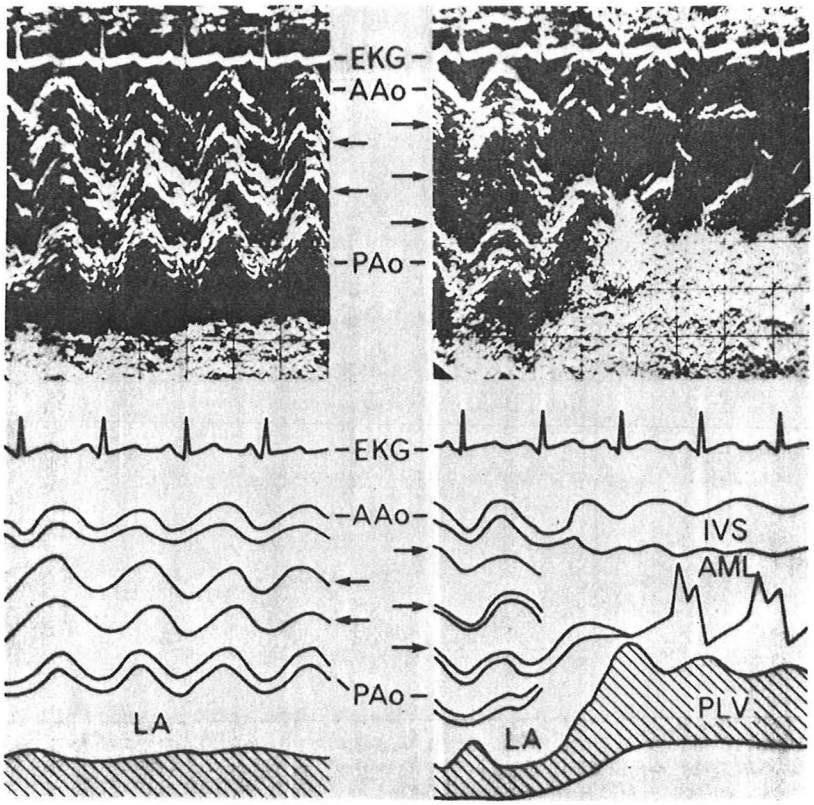

Figure 15. Aortic dissection. Echocardiogram demonstrates multiple parallel discrete echoes in the aorta (arrows). The posterior aortic wall (PAo) is diplaced posteriorly into the left atrium (LA) on the roll-off view of the aorta to the anterior leaflet of the mitral valve (AML) and is not contiguous with this leaflet. AAO = anterior aortic wall; EKG - electrocardiogram; IVS - interventricular septum; PLV - posterior left ventricular wall (150).

Echocardiography can diagnose dissections , however, there are many false positives and false negatives which must be considered. False negatives would occur if the dissection does not extend into the anterior or posterior wall of the aortic root.

Roentgenology

On posteroanterior and lateral chest films several findings may be seen (12,38,42,153).

TABLE 5
X-ray Findings in Dissection

1. Superior mediastinal widening
2. Widening of the distal aortic knob past the origin of the left subclavian artery
3. Double shadow (calcification well inside aortic shadow)
4. Disparity in size of ascending and descending aorta
5. Flattening of aortic shadow on lateral film
6. Bulge of aorta
7. Pleural effusion (usually left)
8. Progressive enlargement of aorta
9. Cardiomegaly
10. Deviation of trachea to the right
11. Normal chest film

The best method of making a diagnosis is by angiography. Many different approaches may be used. Venous angiography by injection in the right atrium or pulmonary artery is relatively safe with minimal chance of rupture during angiography but is frequently not diagnostic with false positives and negatives (154,155). Direct aortography via the femoral, axillary, or brachial routes with positioning of the catheter in the ascending aorta or arch is preferable (156-162). Direct aortography has the best diagnostic accuracy but still has many false positives and false negatives.

Direct signs of dissection such as double channels, linear radiolucency, entry, or re-entry sites make diagnosis of dissection almost certain. Indirect signs such as compression of the true lumen, thickening of the aortic wall, ulcer-like projections, abnormal catheter position, and extravascular extravasation are suggestive but not diagnostic of dissection. Aortic insufficiency is non-specific (41).

TABLE 6
Aortographic Findings in Aortic Dissection

I. Direct Signs
   1. Double channels
   2. Linear radiolucency (torn or separated intima)
   3. Entry
   4. Re-entry

II. Indirect Signs
   1. Compression of aortic (true) lumen by (false) lumen
   2. Thickening of aortic wall (greater than 5-6 mm, normally 2-3 mm)
   3. Ulcer-like projection
   4. Abnormal catheter position
   5. Extravascular extravasation

III. Non-specific Finding
   1. Aortic regurgitation

IV. Branch involvement (see Fig. 10, 11, 12 on pages 14 and 15)

It should be pointed out that there are certain pitfalls as outlined in Table 7 (41):

Table 7
Pitfalls in Aortographic Diagnosis of Aortic Dissection

I. False Negative
   1. Simultaneous and equal opacification of true and false channels
   2. Inappropriate projection
   3. Acute external rupture

II. False Positive
   1. Thickening of aortic wall (due to clotted aneurysm, mediastinal hematoma, atherosclerosis, neoplasm, accumulation of fat, aortitis)
   2. Layering of contrast material (due to aortic insufficiency or Valsalva effect)

When angiography is performed, it is essential to diagnose the entry site, the re-entry site if present, the exact extent of dissection, and the vessel involvement. At times it is difficult to identify if you have your catheter in the true or false lumen. Differentiation may be aided by the factors in Table 8 (41).

TABLE 8
Differentiation between True Lumen and False Lumen

	True Lumen	False Lumen
Location	"normal"	Anteriolateral in ascending aorta Posteriosuperior in aortic arch Posteriolateral in descending abdominal aorta
Pressure	Systemic	Low
Flow	Rapid	Slow
Size of lumen	Often compressed	Often wide
Blood supply	Coronary, intercostal, lumbar	Usually not coronary, intercostal, lumbar

GF was a 71 year-old hypertensive black man who presented with a Type I dissection. His chest film and angiograms are shown in Figures 16, 17, and 18.

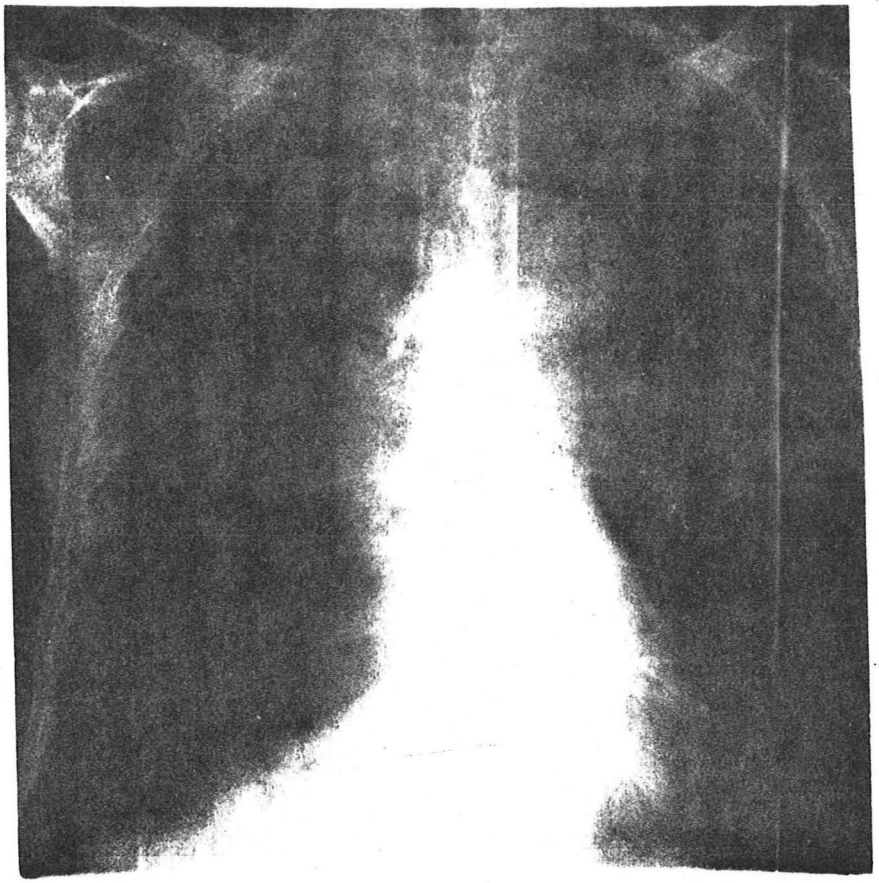

Figure 16. PA chest film. Note the widened superior mediastinum.

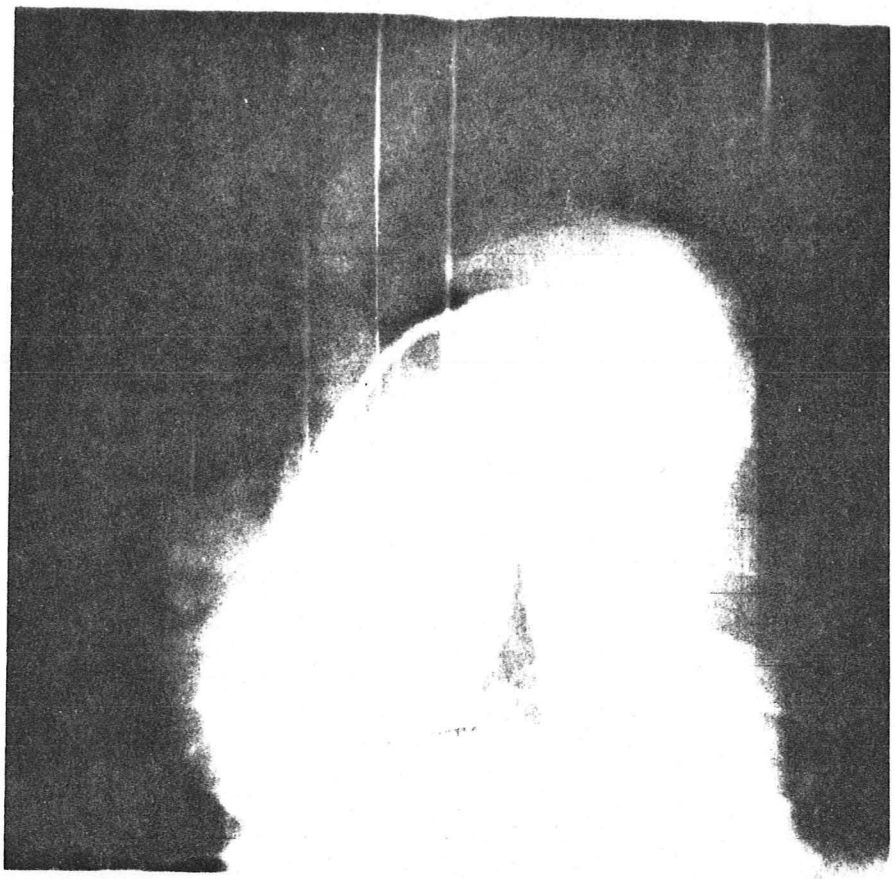

Figure 17. Injection into true lumen. Note the double channels in the ascending aorta and radiolucent lines in the aortic arch. Also note that the aortic valve and innominate arteries are not visualized.

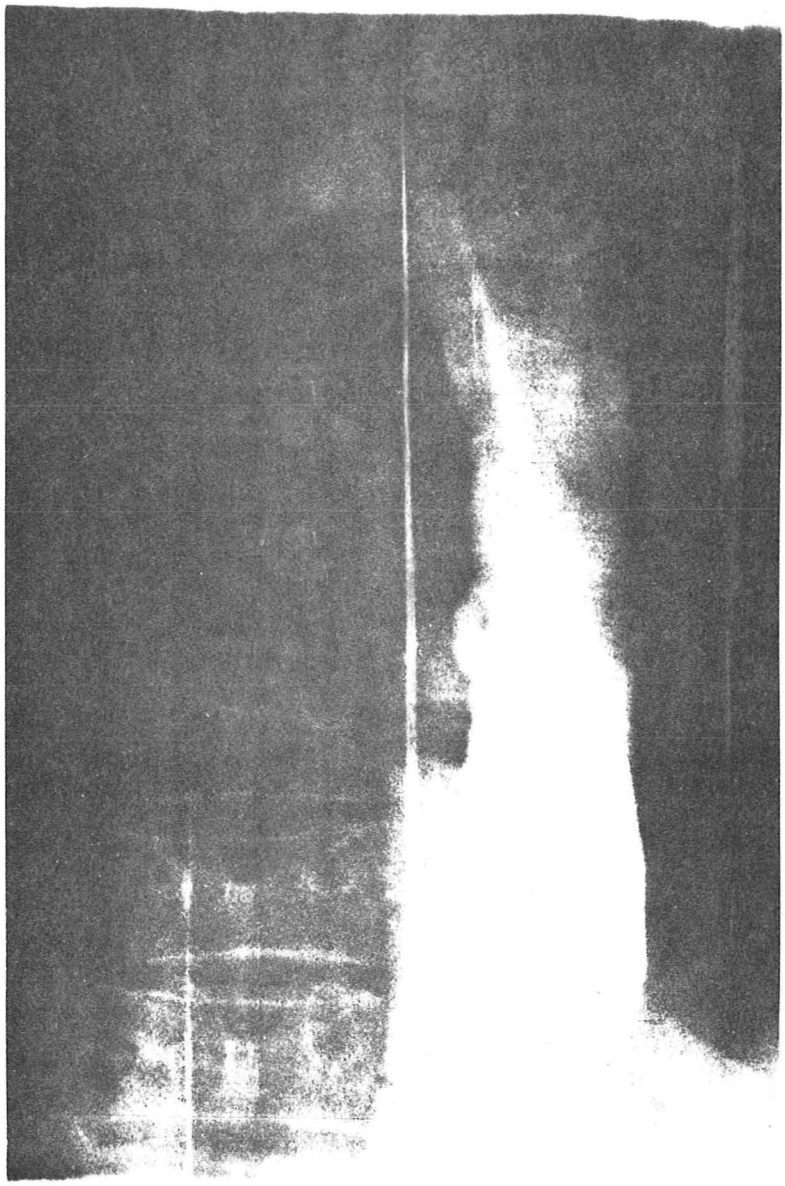

Figure 18. Injection into the false lumen. Note the faint opacification of the true lumen with compression of the true lumen forming 2 channels. Note also the radiolucent intimal flap between the 2 vessels and the ulcer-like projection into the true lumen.

IY was a 50 year-old white woman with left shoulder pain and no pulses in her left arm. She had a history of hypertension. Her chest film and angiogram are shown in Figures 19 and 20.

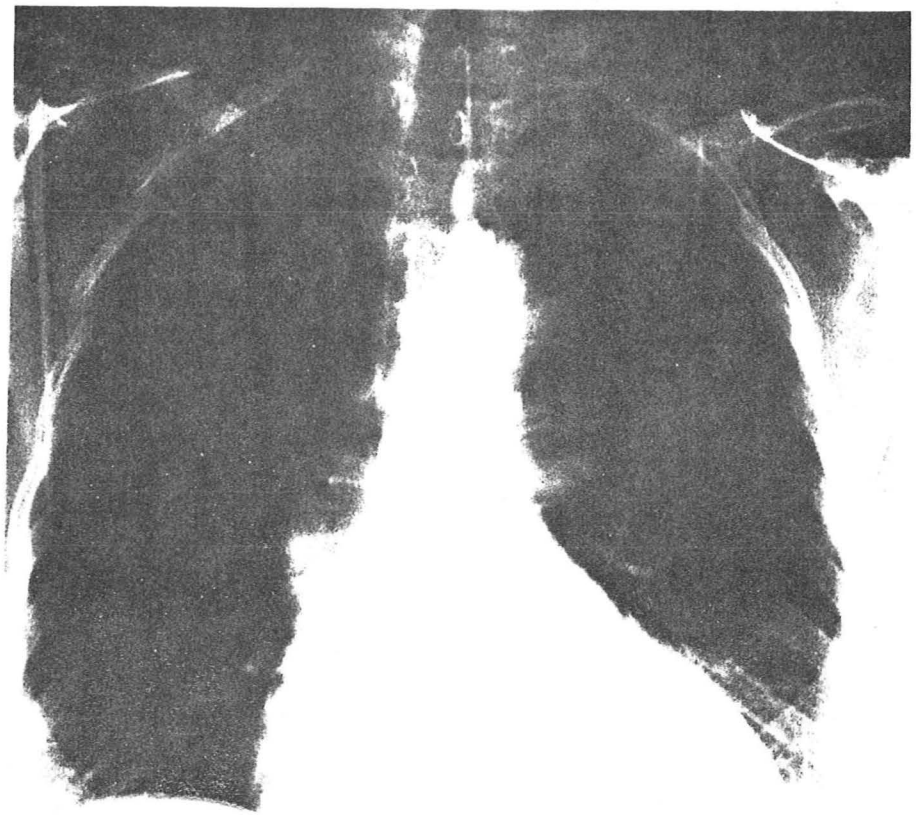

Figure 19. PA chest film. Note the ascending aortic widening which is not marked. There is a left pleural effusion.

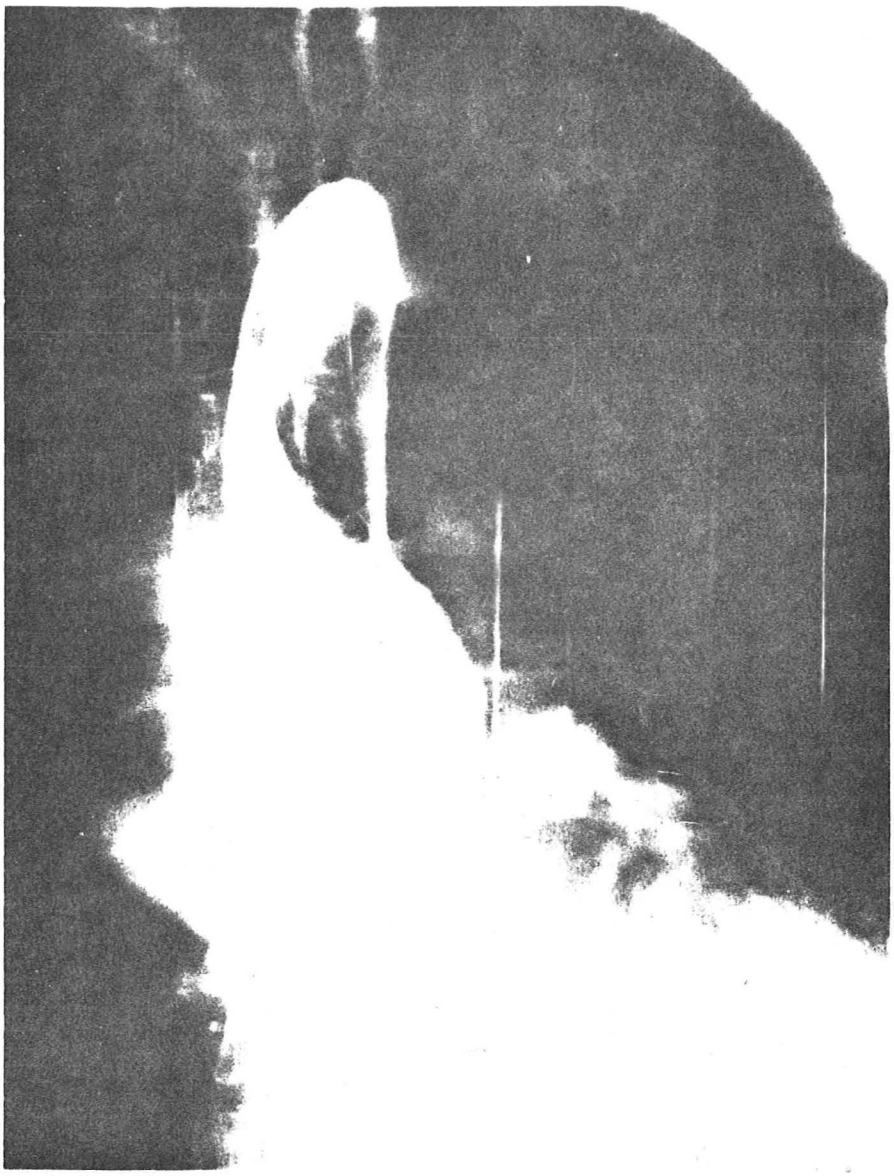

Figure 20. Ascending aortic injection. Note the two lumens with collapse of the true lumen. Note the numerous radiolucent lines representing intimal flaps. Involvement of the great vessels by intimal flaps is also seen. There is aortic regurgitation and extravasation of dye into the pericardium.

EA was a 59 year old black woman with severe hypertension who entered with an increase in aortic size; there were no symptoms. X-rays are shown in Figures 21, 22, and 23.

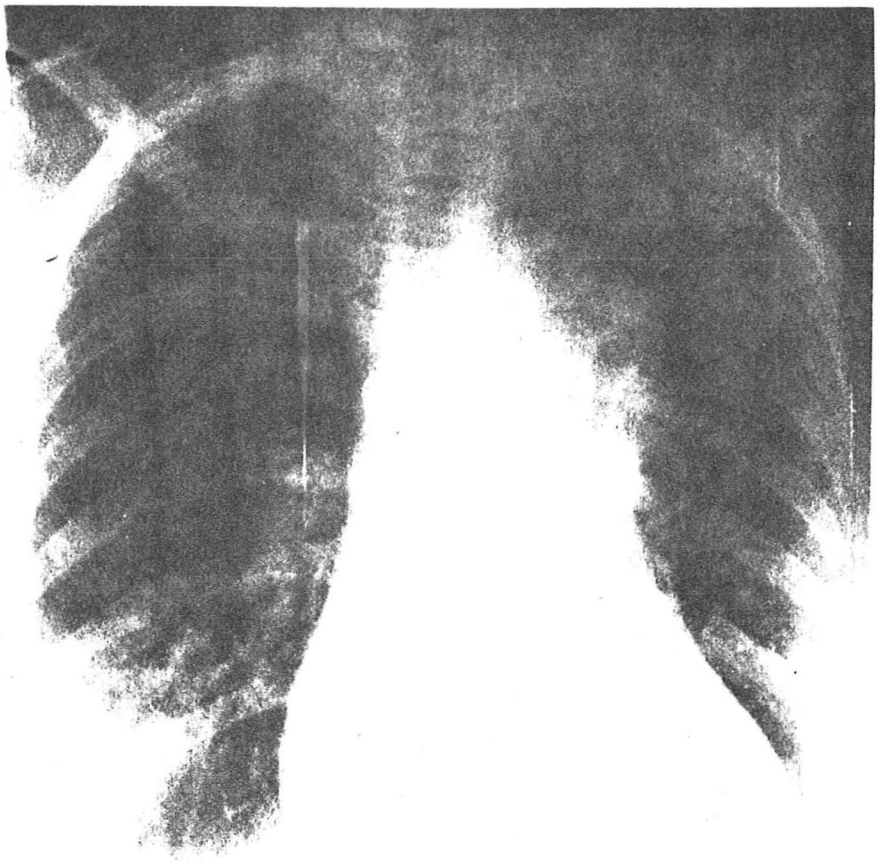

Figure 21. PA chest film. Note the widening of the mediastinum and the disparity between ascending and descending aorta.

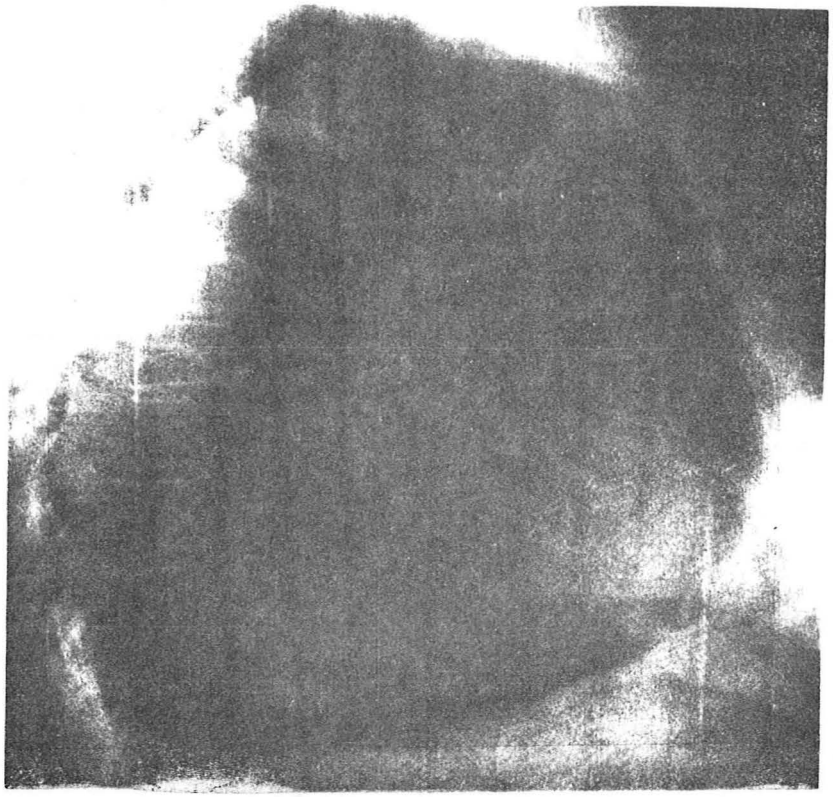

Figure 22. Lateral chest film. Note the flattening of the aorta and bulge at the top of the arch.

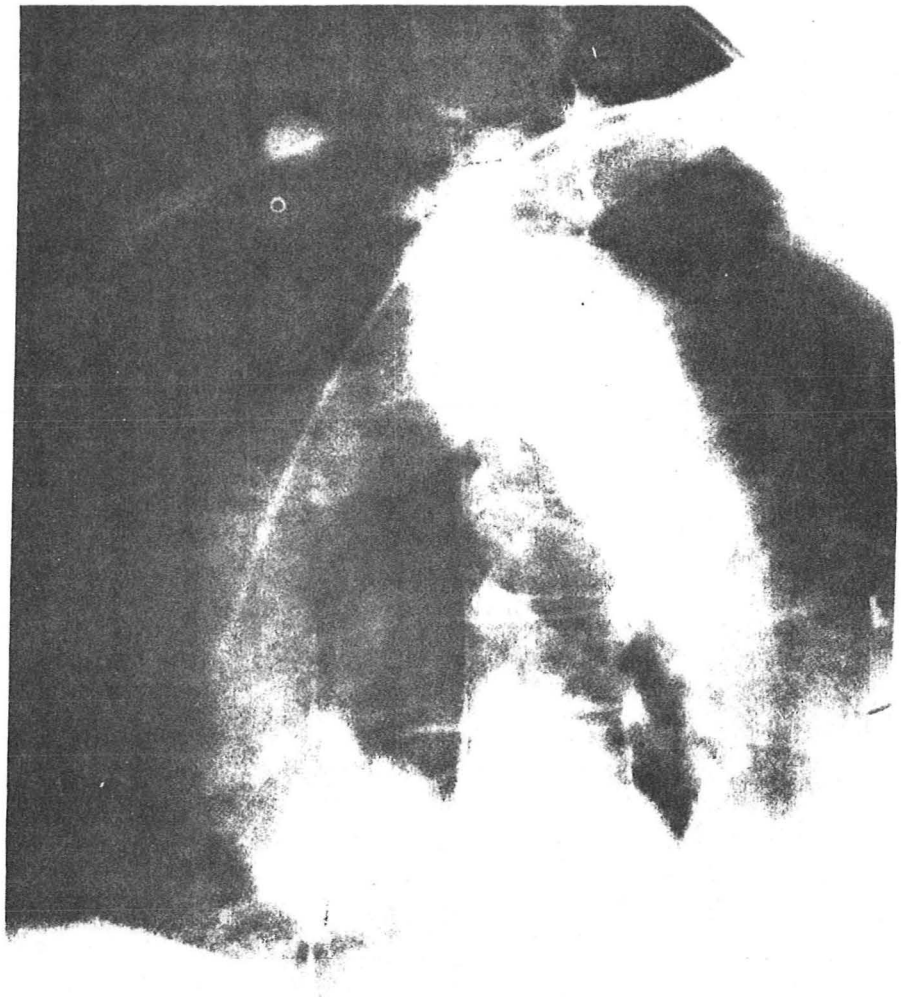

Figure 23. Aortic root injection. Note dissection in descending aorta. There is a radiolucent line which can be seen at the top of the aortic arch and the ragged tear can be seen. The false lumen is clotted without opacification. The true lumen appears slightly narrowed.

Differential Diagnosis

TABLE 9

Syndromes Frequently Confused with Dissection

1. Myocardial infarction
2. Cerebrovascular Accident
3. Pulmonary embolus
4. Rupture of sinus of valsalva
5. Acute surgical abdomen

Acute aortic dissection should always be considered when one of the diseases mentioned above is being considered.

Prognosis

Untreated aortic dissection is a rapidly progressive disease in which 95% of the patients reach the emergency room alive but 50% die within 48 hours. For this reason diagnosis must be made promptly and therapy instituted immediately to prevent this outcome. A review of the mortality in untreated patients is shown in Figure 24:

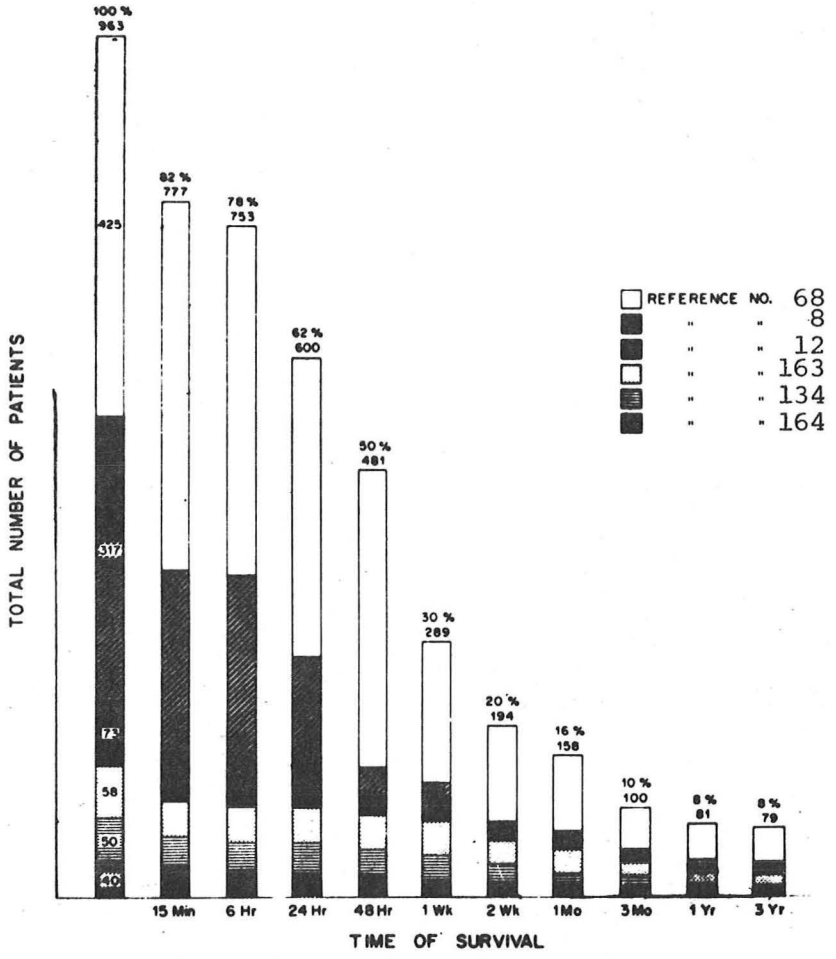

Figure 24. Untreated acute aortic dissections: patient survival (84).

However when dissections are considered by location there is a disparity between types of dissection with descending aortic dissections doing much better than ascending aortic dissections. Surgery improves greatly the survival over untreated patients but only questionably increases survival over medically treated patients without complications. Figures 25 and 26 show the surgical and untreated survival statistics for ascending aortic dissections and descending aortic dissections respectively:

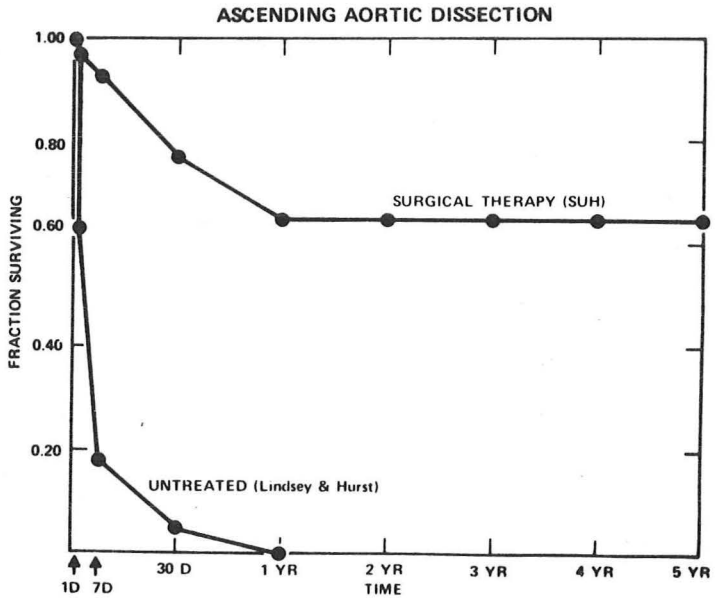

Figure 25. Ascending aortic dissections. Survival in surgically treated and untreated patients (165).

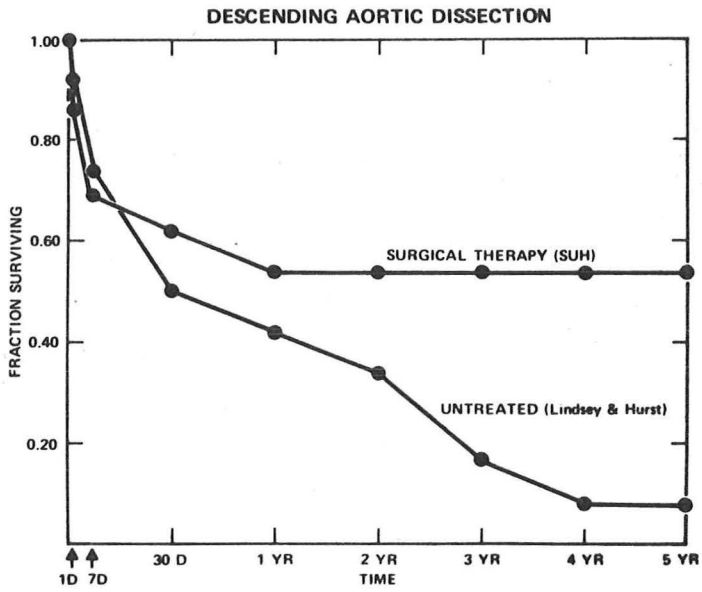

Figure 26. Descending aortic dissections. Survival in surgically treated and untreated patients.

Treatment

In 1965, Wheat (10) revolutionized the treatment of dissection by introducing medical management designed to reduce the blood pressure and decrease the contractility of the heart; this greatly increased survival from 20% to the 50-60% range. More recently Wheat (11), Anagnostopoulos (6) and others have refined this therapy and combined it with surgical intervention at appropriate times to further increase survival. Anagostopoulos (6) has developed an ABC classification of dissections; A being any ascending or arch involvement regardless of where the entry tear is, B being any dissection limited to the descending aorta, and C being patients who are inoperable due to other diseases, end-organ damage, or moribund. He subdivides A and B into complicated and uncomplicated. Table 10 shows this classification.

TABLE 10

The "ABC" Classification of Acute Dissections (6)

Class	Treatment
A. Involve ascending aorta	
A_1 with complications	Medical, earliest surgery
A_2 without complications	Medical, planned surgery
B. Do not involve ascending aorta	
B_1 with complications	Medical, early surgery
B_2 without complications	Medical, ? elective surgery
C. Inoperable	? Medical

Complications: Indications of Surgical Therapy

1. Resistant shock
2. Overwhelming aortic insufficiency
3. Saccular aneurysm
4. Tamponade
5. Myocardial ischemia
6. Reversible central nervous system syndromes
7. Pulmonary artery obstruction
8. Right bundle branch block
9. Resistance to antihypertensives
10. Impending rupture on x-ray
11. Carotid obstruction
12. Normotensive
13. Marfan's syndrome
14. Pregnancy
15. Coarctation
16. Arch dissection
17. Failure of medical therapy (pain, anuria)

Indications for Medical Therapy

1. Clotted false lumen
2. No clear cut origin of dissection visible on angiography in the absence of overwhelming aortic insufficiency or tamponade
3. A stable B_2 dissection with previously untreated hypertension and especially serious concomitant pulmonary disease
4. A stable redissection in a patient improperly treated since first dissection
5. Multiple simultaneous dissections
6. Unavailable cardiac surgical facilities
7. Patient refuses surgery

The current protocol used by Anagnostopoulos (6) and Wheat (11) is given below:

"1. As soon as the possibility of dissection is raised, the attending and senior resident of the cardiac surgery service are consulted.

2. An intravenous route (preferably central venous) is established and a solution of 500 mg Arfonad (trimethaphan) in 500 cc D5W is prepared.

3. Following careful physical examination and review of pertinent data, if the original impression of dissection remains plausible, the Arfonad solution is infused. In most patients the initial blood pressure will be elevated and a rate of 50 mg of Arfonad per hour can be safely started. A reduction in the blood pressure is usually seen within 15 minutes. If not, the infusion rate is increased. If there is still no hypotensive effect, additional drugs may be considered. In the absence of heart failure the use of propranolol is a consideration; however, it should probably be withheld. Should the patient be operated on, a negative inotropic agent with effects lasting for hours or days is undesirable.

4. If the diagnosis of an acute dissection is obvious and the patient is slipping into shock with overwhelming aortic insufficiency and/or a reversible cerebral syndrome, then he should be taken directly to the operating room without further delay (116).

5. An electrocardiogram and regular chest x-ray are ordered if the patient is stable while blood is drawn for type and cross-match (8 units of blood), hematocrit, CBC, electrocytes, BUN, creatinine, SGOT, CPK, bilirubin, prothrombin time, and PTT.

6. With rare exceptions, an aortogram is performed next. The cardiologist, anesthesiologist, radiologist, intensive care unit personnel, operating room, and pump team are alerted.

7. Foley catheter insertion follows. The object of blood pressure control is achievement of the lowest possible pressure consistent with mental alertness and urine output of at least 30 cc per hour.

8. With an anesthesiologist, a cardiologist, radiologist, surgeon, and a nurse, the patient is taken to angiography. Movements from bed to table should be as smooth as possible and sudden jerks should be completely avoided. It may take four to six people to move a patient smoothly.

9. While in angiography, vital signs are consistantly monitored and blood gases determined. Metabolic acidosis should be corrected. An echocardiogram may be considered at this point.

10. The best femoral artery is selected and using the Seldinger technique a catheter is inserted as far as is possible without resistance.

With fluoroscopy control it is positioned in the ascending aorta and a hand 'puff' injection of dye follows to confirm its location.

The arterial pressure is monitored from the catheter and should be below 140/90, reduced from the typical 200/110 on admission. The patient is sedated with morphine (5-8 mg IV) and oxygen is administered. He is told to hold his breath and expect a warm flush as the injection proceeds.

11. Interpretation of the angiogram follows.
    a. If the patient has no demonstrable dissection and the findings preangiogram were very suggestive another injection should be done more proximally (at the aortic root or left ventricle) along with delayed films which may visualize a hematoma without intimal tear. Multiple projections may be needed.

    b. If the repeat injection is negative, another diagnosis must be entertained and the patient is taken to the medical intensive care.

    c. If the angiogram confirms a class A dissection, then the patient is taken to the operating room if complications are present (class A_1, Table 10). Needless to say, this is the approach that a large medical center with a stand-by cardiac surgical team can follow. Continuing medical therapy, although feasible, is not desirable. Preferably, there should be follow-up x-rays of the abdominal aorta so that the complete extent of the dissection is known prior to establishment of bypass.

    d. If the angiogram demonstrates a class B_2 dissection with involvement limited proximally by the subclavian artery and no complications, then intensive care unit and medical management follow. In rare instances a class B_1 acute dissection may be present because of complications on admission. Early surgery is favored by us in such a circumstance.

    e. Patients who are in class A_2 [i.e., without associated complications (Table 10)] can be initially managed medically and within a day or two if stable, operated upon, unless complete experienced cardiac surgical facilities and personnel are available at the outset.

    f. Patients with class A who have question of involvement of the aortic arch should be strongly considered for surgery because tears here should probably be placed with those in class A_1 despite contradictory evidence in the literature (v.i. -surgical princ. -arch).

Variations of this protocol will occur according to the availability and experience of the surgeon. Deferring the decision to operate for a few hours or days under close observation in the patient who responds well to medical management cannot be criticized since the early results of successful medical management in the absence of shock, Marfan's, coarctation, or pregnancy are acceptable. In fact, our review of

questionnaire patients shows that the medical and surgical management differ very little in the over-all 30-day survival! The problem is that it is not possible to predict which class A dissections will remain stable and which will not after a few hours, days, or months after initial stabilization. At the University of Chicago, practically the only class A acute dissections that would not be taken to the operating room would be the rare cases presenting with an acute history and a *clotted lumen* without any signs of aortic insufficiency or cerebral involvement. However, initial medical management even in A_1 dissections on the way to transfer to a bigger center is certainly acceptable therapy, since the alternative of attempting repair in an unprepared center would yield poor results. Basically, then, we adopt Wheat's philosophy (11) that different criteria should be used in determining the plan of action for the patient who presents to a community hospital without open heart facilities and for the patient presenting to a center where facilities for open heart surgery are available around the clock. Medical therapy is easily standardized, but surgical therapy depends on the skill and experience of individuals. This should not be *misinterpreted* to mean that patients can be optimally treated by medical therapy alone. We feel strongly that patients with A_1 dissection or with a true class B_1 are preterminal and require surgical therapy. There are exceptions where such patients have survived medical management, however, just as there are exceptions of perforated duodenal ulcers and appendicitis surviving medical management.

12. For nonsurgical patients, particularly class B_2 and C, intensive care unit treatment follows according to the "medical therapy" protocol.

13. The obvious surgical patients (class A_1 and B_1, Table 10) are taken to the operating room and carefully transferred to the operating table. An arterial cannula, central venous pressure line, and a large bore blood cannula are inserted. The patient is given morphine and succinylcholine and intubated. Antihypertensive therapy is continued, and the chest, abdomen, and both inguinal areas are shaved, prepped, and draped. (6)

The timing for the surgical and medical interventions is shown in Table 11.

TABLE 11

Timing Of Therapy for Dissection (6)

Class	General Operability	Presentation	Treatment 0-4 hours	4-24 hours	1-4 weeks	After 1 month
A_1	Yes	Complicated[a]	Drugs	Surgery	Drugs	Drugs
A_2[b]	Yes	Uncomplicated	Drugs	Drugs	Drugs→surgery	Drugs
B_1	Yes	Complicated[a]	Drugs	Surgery	Drugs	Drugs
B_2[b]	Yes	Uncomplicated	Drugs	Drugs	Drugs, ? surgery	Drugs, ? surgery
C	No	Complicated[a]	?Drugs	?Drugs	?Drugs	?Drugs

[a]One or more of the following: resistant shock, overwhelming aortic insufficiency, saccular aneurysm, tamponade, myocardial ischemia, reversible central nervous system syndromes, pulmonary artery obstruction, right bundle branch block, resistant to antihypertensives, impending rupture on x-ray, carotid obstruction, normotensive, Marfan's, pregnancy, coarctation.

[b]Includes all clotted false lumens

Using these criteria strictly Anagnostopoulos has had very good success in his small series.

TABLE 12

Survival of Last 20 "Operable" Patients since January 1972; Adherence to "ABC" Treatment-Oriented Classification (6)

Class	No. of Patients	Treatment Medical	Treatment Surgical	30-Day Survival Medical	30-Day Survival Surgical	Late Survival Medical	Late Survival Surgical
A_1	11	-	11	N.A.[a]	10/11 (91%)	N.A.	8/11 (73%)
A_2	3	3	-	3	N.A.	3[b]/3	N.A.
B_1	3	-	3	N.A.	3/3	N.A.	3/3
B_2	3	3	-	3/3	N.A.	3/3	N.A.
Total	20	6	14	6/6 (100%)	13/14 (93%)	6[b]/6 (100%)	11/14 (79%)

[a]Not applicable.

[b]One patient may require surgery.

Anagnostopoulos (6) reviewed the literature in patients treated in a relatively aggressive manner, i.e., most patients with complications getting early surgical intervention. Table 13 shows acute (1 month) and chronic (1-5 year) survival as well as redissections.

TABLE 13

Survival in Aortic Dissections (6)

Q = 269
L = 280 Ratio of number of patients surviving to total number in subgroup

	Class A	Class B	A + B
Surgery			
Acute survival (literature)	74/109=68%	70/106=66%	144/225=67%
Chronic survival (questionnaire)	78/110=71%	54/90=60%	132/200=66%
Redissections (questionnaire)	10/110= 9%	2/90= 2%	12/200= 6%
Medicine			
Acute survival (literature)	24/31=77%	28/34=82%	52/65=80%
Chronic survival (questionnaire)	20/40=50%	22/29=76%	42/69=61%
Redissections (questionnaire)	5/40=12%	1/29= 3%	6/69= 9%
Total (Medical and Surgical)			
Redissections (questionnaire)	15/150=10%	3/119= 3%	18/269= 7%
Redissections (literature and questionnaire)			64/549=11%
Total number of patients from literature review			= 280
Total number of patients from questionnaire			= 269
Total			= 549

It is important to remember that all high risk patients received surgery. The acute (1 month) survival appears to be slightly better with medical therapy than with surgical therapy; however, surgical therapy is probably weighted due to high risk patients. Class A patients with surgery, however, have a much better survival chronically (77% vs 50%). Class B patients have a slightly, but not significantly, better survival with medical management. Hence, the statistics support Anagnostopoulos (6) and Wheat (11) in that any Class A dissection should have surgical repair; within 24 hours if complicated or at 3-4 weeks if uncomplicated. Class B patients should be treated surgically within 24 hours if complicated and medically if uncomplicated. A class B dissection treated medically should be reangiogramed at 6-8 weeks to see if the true lumen has closed off; if the false lumen is still patent then it should be surgically closed (6,11,166).

The initial drug of choice appears to be trimethaphan (Arfonad) because of its potent action, rapid onset, and negative inotropic effect. Arfonad 500 mg is added to 500 ml D5W and an initial drip rate of 20 microdrops/minute is used. Due to tachyphylaxis a second drug must be added fairly quickly.

Diazoxide (Hyperstat) and sodium nitroprusside (Nipride) have been used by some authorities. Though they have a potent action and rapid onset, they also have reflexively induced tachycardia and an increased inotropic effect which are detrimental (84). This tachycardia and increased inotropic effect can only be partially blocked by propranolol (Inderal). It should also be mentioned that several authorities (11,84) prefer that propranolol not be used if there is a chance that the patient might go to surgery.

Reserpine IM and aldomet IV can be added to the Arfonad to maintain control when tachyphylaxis appears. Blood pressure should be kept below 140/90 and preferably between 90 and 100 mm Hg systolic if urine flow (30u/hour), mentation, and cardiac function permit.

As soon as the blood pressure is initially controlled angiography should be performed unless the patient is moribund or inoperable. The question of acute myocardial infarction is difficult. If there is ischemia but no definite infarction, then they should have angiography. If they have infarction and no other major complication, medical management with delayed (2-3 week) angiography might be a rational approach though there is no definite evidence on this point.

On the second through the fifth day oral agents are added to the regimen. The best agents appear to be Serpasil, Aldomet, quanethidine, and propranolol as these antihypertensives do not

cause tachycardia or increases in contractility. All patients should be maintained on propranolol 120 to 160 mg/day.

If the patient is to be treated medically he should have repeat angiograms at 6 weeks to be sure the false lumen is closed and at 1 year to be certain that the aorta is not expanding. Propranolol and antihypertensives should be continued forever.

All patients having surgery should also be controlled on propranolol and antihypertensives.

Conclusion

Aortic dissection is a catastrophic illness that needs prompt (in minutes) therapy with an aggressive approach with both medical and surgical means if we are to increase survival in this group. Dissection is not a rare disease but one that is rarely diagnosed. Greater attention to the possibility of dissection is manditory.

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