Observations on the treatment of dissection of the aorta

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Summary

The results are presented of treatment in twenty-three patients with dissection of the thoracic aorta, in four of whom it was acute (less than 14 days' duration), and in nineteen chronic (more than 14 days' duration). Sixteen patients had Type I and II dissection (involving the ascending aorta) and five Type III (descending aorta at or distal to the origin of the left subclavian artery); in two, dissection complicated coarctation of the aorta in the usual site. Thirteen patients had aortic regurgitation.

Three of the patients with acute dissection were treated medically; two, both with Type I dissection, died, and the third, with Type III, survived. The remaining acute patient was treated surgically and also died.

Of the patients with chronic dissection, eight were treated medically and eleven surgically. None of the medical group died in hospital; three died between 3 months and 1 year, and five have survived from periods of 12-72 months. Eleven patients with chronic dissection were treated surgically; four died in hospital at or shortly after operation; and the remaining seven lived for periods of 12-84 months.

The presentation, indications for surgical treatment and results are discussed. It is concluded that surgical treatment of chronic dissection may carry a higher initial mortality than medical, but that there may be slightly better overall long term results in the former. As this series was not selected randomly, because patients with complications were selected for surgery, and there are only a few patients in each group, the results do not permit a firm conclusion regarding the relative merits of medical and surgical treatment.

It is suggested that all patients should initially be treated medically but that surgical treatment should be considered if the dissection continues, if aortic regurgitation is severe, if an aneurysm develops or

enlarges, if cardiac tamponade develops or there is evidence of progressive involvement of the branches of the aorta.

Attention is drawn to the important syndrome of chronic dissecting aneurysm of the ascending aorta with severe aortic regurgitation which requires definitive surgical treatment and aortic valve replacement.

The importance of adequate visualization of the origin and extent of the dissection as a preliminary to surgical treatment is stressed.

THE management of patients with dissection of the aorta is still a matter of controversy. The surgical treatment of this condition, best exemplified by DeBakey *et al.* (1965), has produced a remarkable reduction in mortality. The medical treatment and rationale, advocated by Wheat *et al.* (1965, 1969), has likewise produced striking survival figures, particularly in the acute patients. In spite of these good reports, both surgical and medical management have often tended to produce poorer results and the selection of the best therapy for any individual patient is often difficult.

The purpose of this paper is to present the clinical features and results of treatment in twenty-three patients with dissecting aneurysm of the aorta and, in the light of this experience and that of others, to outline some of the prognostic features and indications for medical and surgical treatment.

The patients

Twenty-three patients with dissection of the aorta admitted to Hammersmith Hospital during the period 1962-1972 were studied. Patients were included only if the diagnosis was by aortography (fifteen patients), at operation (in seven patients) or at post-mortem (one patient). There were seventeen men and six women with an age range of 37-66 years.

The dissection was considered acute if the symptoms lasted less than 14 days (four patients), and

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chronic if they lasted more than 14 days (nineteen patients). Since the diagnosis was definite in all patients, classification into one of the three main types (DeBakey *et al.*, 1965) was possible.

Type I. Dissection involving the ascending aorta, aortic arch and extending distally for varying distances (thirteen patients).

Type II. Dissection limited to the ascending aorta (three patients).

Type III. Dissection originating at or distal to the left subclavian artery and extending distally for varying distances (five patients).

Two patients in whom the dissection was related to coarctation of the aorta have not been classified in this way but remain in the series.

Even with aortography and the appearance at operation, distinction between Types I and II can be very difficult, but the absence of involvement of the major vessels to the arms and neck suggests Type II. For practical purposes it is vital also to consider involvement of the aortic valve, which is a complication that strongly influences the method of treatment adopted.

Aetiology

Systemic hypertension

The commonest aetiological factor, systemic hypertension, was present in thirteen of the twenty-three patients. It was noted in all five patients with Type III dissection. One patient developed malignant hypertension while under medical treatment for his dissection. Hypertension appears to be more frequent in Type III than in Types I and II dissection (Lindsay and Hurst, 1967).

Disease of the aortic wall

In four patients there was medial deficiency of the aortic wall of the Marfan type, three of these having had Type II dissection. Only one of the four patients showed the classical physical features of Marfan's syndrome, although another was tall and had pectus excavatum. In one patient, the medial coat of the aorta showed cystic necrosis.

Coarctation of the aorta

Two patients had coarctation: post-ductal in one, with dissection commencing proximal to the coarctation, and pre-ductal in the other, with the intimal tear distal to the coarctation.

In seven patients no obvious causative factor was found, but in one of these there was a significant past history of chest trauma, while another had a family history of aortic rupture and a third had bacterial endocarditis on a ruptured aortic valve cusp, and vegetations were also seen at the edge of the dissection.

Clinical features

Chest pain

The commonest presentation was with chest pain (in twenty-one of the twenty-three). The description of the pain varied but it was usually very severe, and radiated sometimes to the back between the scapulae, to the arms, and with progression of the dissection to the abdomen and even down the thighs. In three patients with Type I dissection, the initial pain was soon followed by typical angina on effort. Persistence of pain was an unfavourable sign, indicating extension of the dissection and, conversely, disappearance of the pain suggested a stage of stabilization.

Inequality of arterial pulses and low back and abdominal pain

Inequality of the upper limb pulses and aortic regurgitation were features of Type I and II dissections, whereas backache, abdominal pain and hypertension were commoner in Type III.

Aortic regurgitation

There was aortic regurgitation in thirteen patients, of whom ten had Type I and three Type II dissections. A Venereal Disease Research Laboratory test, Rose-Waaler and Reiter protein complement fixation tests were negative in these patients.

Electrocardiography

The electrocardiogram was helpful in a negative way in that only one patient showed a pattern of cardiac infarction. The commonest findings were non-specific T-wave changes and/or left ventricular hypertrophy.

Radiology

The most frequent abnormality on chest radiographs was dilatation of the ascending and/or arch of the aorta. There was calcification in the arch or ascending aorta in two patients. Two patients with Type III dissection had a left basal plural reaction.

Treatment and results (Tables 1-3)

The decision to treat a patient medically or surgically was not made by random selection but was determined by the judgment of the attending physician or surgeon at the time. In this respect, and because of the varying causes of dissection, the presence of aneurysm in some patients but not in others, the small numbers involved, and the selection of patients for surgery because of complicating factors, the medical and surgical groups are not comparable, but their management and the results of treatment offer some useful lessons. Of the twenty-three patients, eleven were treated medically and twelve surgically (Tables 1 and 2).

There were four patients with acute dissections,

TABLE 1. Summary of patients treated surgically

Patient	Age	Sex	Type	Duration of survival after operation	Cause of death
AD	56	M	Postductal coarctation	10 days	Pulmonary oedema and cerebral infarction
HW	56	M	I Acute	Operative death	Intractable ventricular fibrillation
BL	50	M	I Chronic	6 days	Pulmonary oedema and renal failure
SA	54	M	III Chronic	1 month	Ruptured graft
EJ	62	M	I Chronic	Operative death	Uncontrollable haemorrhage
BJ	54	M	II Chronic	5 years	Alive
BJ	65	M	I Chronic	2 years	Alive
RW	50	M	I Chronic	4 years	Alive
MC	54	F	I Chronic	5 years	Alive
LH	59	M	III Chronic	16 months	Alive
ET	45	F	Postductal coarctation	1 year	Alive
TM	64	M	II Chronic	7 years	Alive

TABLE 2. Summary of patients treated medically

Patient	Age	Sex	Type	Duration of survival	Cause of death
RR	62	F	I Acute	7 days	Extension of dissection
CD	37	F	I Acute	3 days	Extension of dissection
WG	55	M	III Acute	3 years	Alive
BE	58	M	I Chronic	6 years	Alive
LA	66	F	I Chronic	18 months	Alive
HA	66	M	I Chronic	6 months	Alive
SF	38	M	I Chronic	3 months	Rupture into pericardium
CE	61	M	III Chronic	3 years	Unknown
GW	59	M	II Chronic	7 years	Unknown
SA	41	F	I Chronic	1 year	Extension of dissection
HP	48	M	III Chronic	3 months	Rupture into mediastinum

three of whom were treated medically and one surgically. Of the three who were treated medically two died, but probably had not received what is now considered optimal management as the regime of intensive hypotensive therapy combined with negative inotropic agents was not well established at that time. Both these patients died from the effects of progressive extension of the dissection and one became paraplegic owing to spinal cord infarction a few hours before death. In the one acute patient who was treated surgically the dissection was associated with a coarctation, and he died 10 days after operation.

The remaining thirteen patients all had chronic dissections and eight of these were treated medically and eleven surgically.

Medical treatment

The decision to treat medically was based on the relatively stable nature of the dissection up to that time, the lack of ischaemia affecting a major organ or limb, the absence of significant aortic regurgitation, and the relatively high risks of early surgery. Six of these patients had a dissection originating in the ascending aorta, five having mild to moderate

aortic regurgitation during the course of their illness. Medical therapy in all patients with chronic dissection was principally one of bed rest with close supervision to detect the development of any complications or increase in the size of the aneurysm, and a hypotensive regime usually with the addition of

TABLE 3. Survival of patients with dissecting aneurysm

	Number of patients	Hospital deaths less than 3 months	Late deaths 4-12 months	Late survivors 12 months or more
Acute and chronic				
Medical group	11	2	3	6
Surgical group	12	5	0	7
Total	23	7	3	13
Chronic only				
Medical group	8	0	3	5
Surgical group	11	4	0	7
Total	19	4	3	12
Acute only				
Medical group	3	2	0	1
Surgical group	1	1	0	0
Total	4	3	0	1

propranolol. Three out of the eight patients with chronic dissection treated medically died at intervals of from 3 months to 1 year from a further extension of the dissection. Two others died at 3 years and 7 years respectively after the onset of the dissection of unknown and probably unrelated causes. Three others are alive at periods of from 5 months to 6 years since the dissection. The mean duration of survival in this group was 29 months.

Surgical treatment

Four of the eleven patients with chronic dissection treated surgically died soon after surgery. All the other seven patients are alive and well at periods from 1 to 7 years after operation. The mean duration of survival in the surgical group since the dissection was 49 months.

The indications for surgery in the eleven patients who were operated upon were various complications, namely, marked aortic regurgitation in seven (with endocarditis of the aortic valve in one), a large aneurysm or increase in size of the aneurysm in three and the presence of a dissection in relation to a coarctation in one. In eight of these patients the dissection originated in the ascending aorta. The operation consisted in most patients of resection of as much of the aneurysm as possible, together with the initial tear, and insertion of a prosthesis in its place. In addition, aortic valve replacement with a Starr-Edwards prosthesis was carried out in seven patients. Aneurysms of the aortic root were treated by replacement of the aneurysm with a prosthesis, aortic valve replacement and re-implantation of the ostia of the coronary arteries, with wrapping of the prosthesis (which acted as an inlay graft) with the redundant aorta. Two patients (B.J. and R.W.) who required this radical operation have already been reported (Singh and Bentall, 1972).

Case reports

The following two patients illustrate the clinical features of Types I and III dissections.

Case 1

W.G., a 55-year-old man had been hypertensive for 6 years, being treated with bethanidine; he was admitted to the Hospital on 29 November 1968, with a 2-hr history of severe tearing pain in the left lower chest and upper abdomen, and vomiting.

On examination he was alert, in sinus rhythm and the blood pressure was 150/80 mmHg. The left subclavian and brachial pulses were weaker than those on the right; all other peripheral pulses were palpable. There were no murmurs over the chest wall or great vessels. The epigastrium was tender.

He remained oliguric for about 24 hr and then began to pass urine. The electrocardiogram showed

slight ST depression in the left praecordial leads. The chest radiograph showed a very wide thoracic aorta and a pleural reaction at the left base. His ESR was 79 mm in the first hour, the blood urea 135 mg/100 ml, serum amylase 65 units, and lactic dehydrogenase 285 i.u.

A provisional diagnosis of dissection of the aorta was made and an aortogram was performed by the Seldinger technique via the right femoral artery. This showed 90% occlusion of the left renal artery and a smooth narrowing of the aorta below the superior mesenteric artery. The pyelogram showed some excretion of dye by the left kidney, and a normally functioning right kidney.

Progress. About one hour after the aortogram, the blood pressure rose to 190/120 mmHg. Despite intramuscular reserpine and phenoxybenzamine, the blood pressure remained elevated (170/110 mmHg), so guanethidine was added on the next day. On the third day reserpine and phenoxybenzamine were stopped and he was treated with parenteral guanethidine, methyl dopa and propranolol, which kept the blood pressure at 100/70 mmHg. As he lost his pain and because of arrest of the progress of his dissection, operation was withheld. On the third day of admission, however, the patient complained of pain in his left hip and a murmur was heard over the left femoral artery. He did not deteriorate further and was discharged home on 8 January 1969, on oral hypotensive therapy, with a blood pressure of 100/60 mmHg.

He was re-admitted to Hammersmith Hospital on the 11 April 1969, because of recurrent hypertension and for investigation of attacks of nausea, vomiting and headaches of 1 month's duration. On examination, he was in sinus rhythm and the blood pressure was 200/140 mmHg. All peripheral pulses were palpable and there was still a bruit over the left femoral artery. He had some weakness of the left thigh and numbness over both buttocks. Routine chemical tests on the blood and the plasma electrolytes were normal. The electrocardiogram showed only minor repolarization changes over the left ventricle. The chest radiograph showed further widening of the descending thoracic laterally and posteriorly.

In view of the apparent increase in size of the aneurysm, a further aortogram was carried out on the 11 April 1969. Two injections were made, the first into the ascending aorta, which showed dilatation and good filling of the main arteries arising from the aorta, except for a short filling defect in the innominate artery. The aorta distal to the left subclavian artery was very narrow, being compressed by a large dissecting aneurysm which extended from just beyond the left sub-clavian artery to just above the diaphragm. In view of the extent of the aneurysm it

was decided to continue medical treatment and to improve control of the blood pressure. He was discharged home on bethanidine and propranolol on the 15 May 1969. Since then he has been regularly followed-up at his local hospital where his blood pressure has been kept under good control and there has been no change in the size of the aneurysm over the last 4 years.

Comment. The extensive nature of the dissection, the absence of aortic regurgitation or severe ischaemia of any organ and the good response determined that conservative treatment should be continued; it was clearly important to control the blood pressure and to reduce the force of left ventricular contraction, to reduce the strain on the aortic wall.

Case 2

T.M., a 64-year-old man, was well until 5 April 1963, when, while walking, he experienced palpitations, shortness of breath and tightness in his chest for 15 min. He rested in bed, but 3 weeks later, after he had resumed activity, he noticed shortness of breath and tightness in the chest on exertion. In July 1963 he was found to have severe aortic regurgitation with inequality of the upper limb pulses; the blood pressure was 100/60 mmHg in the right arm and 160/40 mmHg in the left. There was also a palpable difference in the carotid pulses but the femoral pulses were normal. The electrocardiogram showed left ventricular hypertrophy and the chest radiograph showed a markedly dilated aorta. There was no history of rheumatic fever and the Wasserman and *Treponema* immobilization tests were negative. An aortogram showed gross aortic regurgitation and a dilated aorta, but no dissection was seen.

In November 1973 he was referred to Ham-smith Hospital for further investigation. He was a tall man with a pectus excavatum; symptoms and physical findings were similar to those in July 1963. An aortogram was repeated in two planes but did not show any evidence of dissection. In spite of this, it was felt that the clinical picture and history were those of dissection and in view of increasing left ventricular hypertrophy on the electrocardiogram and persistence of symptoms, with severe aortic regurgitation, operation was undertaken under extracorporeal circulation with continuous perfusion of both coronary arteries during the period of aortic cross-clamping. The heart was considerably enlarged, as was the aorta, which was 6 cm in diameter, with a dissecting aneurysm in the ascending portion extending down to the insertion of the aortic cusps and up to the origin of the innominate artery. Two patent channels were present. The aortic valve was removed with the edges of the orifice where the dissection occurred. The lower edge was sutured to the aortic wall. A no. 11 Starr-Edwards prosthesis

was inserted. The post-operative course was uneventful; the patient has been followed regularly since the operation and is now working and feeling well although he developed atrial fibrillation in March 1970.

Comment. This patient illustrated the need for operation in Type I dissection when the aortic valve is involved.

Discussion

In the management of this condition the first step is to distinguish it from other acute illnesses, particularly myocardial infarction, a cerebro-vascular accident, systemic or pulmonary embolism, and an acute abdominal crisis. An experienced clinician can diagnose a large proportion of cases correctly, but a definitive diagnosis by aortography with demonstration of the site of origin and its extent is imperative if surgery is to be considered. The need for further investigation may be greatest in the seriously ill patient who is otherwise a suitable candidate for surgery. The limitations of aortographic diagnosis (Sheeford, Sybers and Weens, 1969) were evident in the present series, where in four patients, the dissection was demonstrated neither by pulmonary artery injection and follow-through nor by retrograde aortography. The dissection, although suspected, was confirmed at operation in seven patients. The difficulty is greatest in the patient who presents with 'lone' aortic regurgitation with no obvious cause, with or without a history of chest pain, and marked dilatation of the ascending aorta seen on the chest radiograph. Aortic regurgitation with dilatation of the ascending aorta, unless the aetiology is clearly known, should arouse suspicion of a localized aneurysm in the ascending portion of the aorta (Keene *et al.*, 1971). There were three such patients in this series. Surgical treatment is needed in this group and thus aortography is imperative.

In any comparison of medical and surgical therapy, it is of importance to consider certain factors, in addition to therapeutic interventions, that can affect prognosis. Thus, dissections originating in the ascending aorta (Type I and II) have been considered to have a worse prognosis than those originating in the descending aorta (Type III) (McCloy, Spittell and McGoon, 1965; Lindsay and Hurst, 1967). The duration of the dissection is another factor, as the highest mortality of any form of therapy is in the first few weeks. Increasing age and hypertension have less effect on prognosis (McCloy *et al.*, 1965). Recently, McFarland *et al.* (1972) have claimed that patients who failed to show a communication between the true and false lumina at the time of their initial angiograms had a significantly better prognosis than those with such a communication. A review of nine angiograms of the

medically treated group in this series showed that five of these had definite opacification of the false channels. Three of them died within 1 year from an extension of the dissection, one died suddenly 3 years later and the other is alive 5 years later. In four patients no double lumen was seen on angiography; two of these are alive at periods of 1 year and 3 years after diagnosis. Of the other two, one died suddenly 7 years later and the other 1 month after diagnosis from rupture into the pericardium.

Compared to the overall mortality of untreated dissecting aneurysms, which ranges from 75% to 80% in the first month (Hurst, John and Kine, 1958; Austin, Buckley and McFarland, 1967), the results of surgical therapy are very impressive. DeBakey *et al.* (1965) reported 179 patients with dissecting aneurysms who were classified according to the type and duration of the disease. Their best results were in Type III dissecting aneurysms, with a mortality of 19% for both acute and chronic cases, whereas in the Type I acute cases in hospital, mortality was around 40%. In their series, about 50% of the patients had chronic dissections of Type III. The best results with vigorous medical therapy were reported by Wheat *et al.* (1969) in sixty-nine patients. Of their patients with acute dissections of the aorta, thirty-six had Type III, and twenty-eight Types I or II. There was an initial overall mortality of 14% and at the end of one year 84% were alive. These are indeed very impressive results but unfortunately not all their patients had the diagnosis confirmed by aortography. Daily *et al.* (1970) reported thirty patients with acute dissections and claimed a mortality of 28% of all aneurysms treated surgically, but a mortality of 20% for Type III treated medically. The experience of most others (Austen and De Sanctis, 1965; Austen *et al.*, 1967; Atlar *et al.*, 1971; Grandin *et al.*, 1967; Liotta *et al.*, 1971) favours surgical treatment.

In the present series there was a high mortality for acute dissection whatever the method of treatment (Tables 1, 2 and 3). Among chronic dissecting aneurysms, which constituted the majority of the series, the initial results of medical therapy seem better, but there is a higher loss from further extension of the dissection. In the surgical group, all with complications of dissection, there is a higher initial mortality, but all seven of the eleven patients who survived the operation are alive and well, with a slightly longer mean duration of survival than the medical group. However, against this must be placed the fact that five of eight medically treated patients survived for one year or more. With dissections that originate in the ascending aorta (Types I and II) which have a poor prognosis, mainly in view of the tendency to severe aortic regurgitation, myocardial ischaemia and cardiac tamponade, medical manage-

ment has been less successful (Wheat *et al.*, 1969; Harris *et al.*, 1967). The mortality of surgical treatment of Types I and II dissections of the acute stage has been reported as 40% (De Bakey *et al.*, 1965), 30% (Liotta *et al.*, 1971) and 28% (Daily *et al.*, 1970). Thus, in dissection of the ascending aorta it is still difficult to decide whether to operate immediately or only after failure of adequate medical therapy or upon the development of complications such as the presence of marked aortic regurgitation, occlusion of a major vessel or increase in size of aneurysm and impending rupture.

The argument that all uncomplicated dissections of the ascending aorta should first receive medical treatment and that surgery should only be considered after failure of medical therapy to arrest the progress of the dissection is based on the high immediate mortality of surgical treatment and the fact that surgery is more practical and safer on an elective basis at a later date when the aortic tissues are less friable than in the acute situation. Against this must be considered the possible continual reduction in mortality of surgical treatment during the acute stage with advancing improvements in technique. Moreover, the failure of medical therapy may render the patient a higher risk for surgery. Thus, the management of aortic dissection, particularly of the ascending aorta, has to be considered individually in each patient.

The numbers in this series are too small for definite conclusions to be drawn, but the higher immediate mortality from surgical treatment than from medical contrasts with the only slightly better figures for long term survival in the surgical group. However, the four patients who died at or shortly after operation might well have died if treated medically and there is little difference in the overall and long term results.

Daily *et al.* (1970) treated all acute dissections medically initially, proceeding to surgery in good risk patients. Medical therapy was continued in all other patients, such as those with no site of origin visualized on aortography, poor general condition, or stable dissection. If the dissection progressed, the aneurysm enlarged or tamponade or aortic regurgitation increased or developed, surgery was performed. Using this policy in thirty-five patients, surgical treatment of Types I and II dissections produced a mortality of 35% (as compared with 67% for medical treatment) but in Type III dissection the reverse was true, the surgical mortality being 28% as compared with 20% for the medical.

The limited experience in this series would suggest that initially all acute dissections should be treated medically with hypotensive drugs and β -adrenergic blocking agents, and that this form of treatment should be maintained unless there is aortic

regurgitation, increase in size of the aorta, tamponade or evidence of further dissection in the form of interference with branches of the aorta.

In chronic dissecting aneurysm, the type of treatment depends basically on the same factors and it is believed that a stable dissection which is not interfering with the aortic branches and is not causing any aortic regurgitation should be treated medically as a definitive rather than as a presurgical policy, and the figures for long term survival would tend to bear this view out. Aneurysms of the ascending aorta with aortic regurgitation demand operation, with insertion of an inlay graft and aortic valve replacement (Singh and Bentall, 1972).

It must be emphasized that medical treatment does not imply 'laissez faire' but is a positive programme of careful reduction of blood pressure to mildly hypotensive levels and the use of β -adrenergic blockade to reduce the force of cardiac contraction. The aim of medical therapy is thus to reduce the systolic systemic blood pressure to between 100 and 120 mmHg provided that a satisfactory level of consciousness and urinary output of 20 to 30 ml/hr is maintained (Malm, 1972). Reserpine may be given i.v. every 3 or 4 hr to provide a smooth reduction in blood pressure but has the disadvantages of bradycardia and a degree of sedation which may interfere with assessment of progress. Alternatively, pentolinium may be given by injection or methyldopa by mouth. In addition, propranolol should be given to reduce the force of contraction of the left ventricle.

It is essential to observe symptoms, in particular, pain; other essentials are repeated examination of all accessible arterial pulses, and frequent radiographs of the chest to look for increase in size of the aorta denoting progressive dissection or a developing aneurysm. Spontaneous disappearance of pain is an important indication that the dissection has been arrested. Aortography should be undertaken as early as possible to delineate the dissection and aid in planning surgical intervention, preferably as soon as the patient's condition is stable, or if it is clear that medical treatment is not controlling the dissection.

Acknowledgments

Professor H. H. Bentall gave valuable advice and we are grateful to him and to Dr C. M. Oakley for permission to include their patients, and to the Technical and Nursing Staffs of the Cardiac Catheterisation and Angiocardiographic Laboratories, and of the Cardiothoracic Surgical Unit.

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