The racing drivers were influenced by two main determinants—competition and danger—both acting in concert during the race but only in an anticipatory sense before the start, yet capable of consistently inducing heart rates exceeding 130/min. Extremely rapid sinus tachycardia of the same order (180–200/min.) was reported in racing drivers by Collins (quoted by Simonson, Baker, Burns, Keiper, Schmitt, and Stackhouse, 1968) and a somewhat slower heart rate (120–160/min.) by Rapp, Dietlein, and Nuttall (1965) over a two-hour race. We have recorded a heart rate of 120–130/min. in the third hour of the 1968 Le Mans 24-hour race, but we could not continue the observations because of a mechanical breakdown of the car. Certain civil airline pilots develop heart rates of 120–130/min. during critical periods of the flight, including take-off, approaching a busy airport, and landing (Howitt, Backwill, Whiteside, and Whittingham, 1965). Much higher rates (200–240/min.) have been documented in oarsmen after a 500-metre race (J. L. Corbett, personal communication, 1968), but tachycardia associated with physical exercise is a separate problem and is merely mentioned in passing.

We have assumed that the tachycardia and electrocardiographic changes of driving are determined by emotion similar to that responsible for such changes during angry discussion (Bogdonoff, Combs, Bryant, and Warren, 1959), discussing problems of personal importance (Wolff, 1950), before examinations (Bogdonoff, Estes, Harlan, Trout, and Kirschner, 1960), and after a sudden fright induced by a pistol shot (Graybriel and White, 1935). The ectopic beats and short bursts of tachycardia we recorded have a similar emotional origin, probably through the intermedium of induced ischaemia. Excess catecholamine secretion appears to be a common denominator in all these situations.

All the racing drivers showed a considerable increase in noradrenaline compared with the tranquil levels, while the adrenaline content greatly exceeded the tranquil value in one driver only. The city drivers showed insignificant and inconsistent changes. None of these drivers developed angina during the drive, though both drivers 1 and 2 showed ST segment depression. Previous workers have shown considerable increases in urinary catecholamine excretion in drivers and passengers (Schmid and Meythaler, 1964).

We do not wish to read too deeply into these few observations. There is clearly a wide variance between the catecholamine response in different racing drivers. Many factors will have to be investigated before the meaning of these findings is understood, including the driver's personality, the intensity of the competitive element in the race, the mechanical behaviour of the car, and the driver's body temperature involving considerable fluid loss into the anti-inflammable underwear. The city drivers, showing much smaller changes in catecholamine levels between the after-drive and "control" samples, were not exposed to anything like the same driving variables as the racing drivers. We present the findings now as preliminary observation which will form the basis for future tests.

We wish to thank the Automobile Association and the Medical Commission on Accident Prevention, who provided financial support; the Ford Motor Company for lending us a Ford Transit Minibus for transporting equipment to the racing circuits; the officials of the 24-Hour Le Mans Race and Dr. Peter Riley and Mr. J. G. W. Marsh, of Marcos Cars Ltd., for assisting us at Le Mans; the officials of the Royal Automobile Club, British Automobile Racing Club, and British Racing and Sports Car Club for helping us at the race meetings; Medical and Industrial Equipment Ltd., and Ernest Turner Electro Instruments Ltd., for assistance with equipment; and many others, including the drivers themselves.

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Recognition and Management of Dissecting Aneurysms of the Aorta

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Summary: The current approach to treatment at St. Mary's Hospital of dissecting aortic aneurysms uses a period of induced hypotension before aortography and consideration for surgical correction. There are 13 survivors of a series of 24 patients treated in this way.

Introduction

Aortic dissection is a lethal condition. The diagnosis, though not always easy, can usually be made on the clinical history and examination, and confirmed by radiology. As the early clinical features of acute dissection may mimic those of cardiac infarction the distinction must be made, especially if anticoagulant therapy is contemplated.

At the present time there is no general agreement regarding the ideal management of dissecting aneurysms (Lindsay, 1969; Wheat and Palmer, 1968). The purpose of this paper is to present our recent experience at St. Mary's Hospital.

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Pathology

The condition affects either sex and is commonly associated with hypertension and a primary degenerative change in the aortic media. Dissection of the media by arterial blood follows splitting of the intima, which usually occurs at one of two sites: either in the ascending aorta just above the aortic valve or in the descending thoracic aorta just distal to the origin of the left subclavian artery. The dissection progresses distally, circumferentially, and proximally under systolic pressure. Rupture of the "outer" aortic wall results in massive internal haemorrhage. Extension of the dissection into the aortic branches or occlusion of the origin of these vessels can produce acute ischaemia of the coronary, cerebral, spinal, visceral, or limb circulation. Spontaneous re-entry of the dissection may follow rupture of the intima distally. The site of the origin, the extent of the dissection, and the pathological complications influence the patient's chances of survival.

The majority of dissections fall into three types (Fig. 1) (De Bakey *et al.*, 1965).

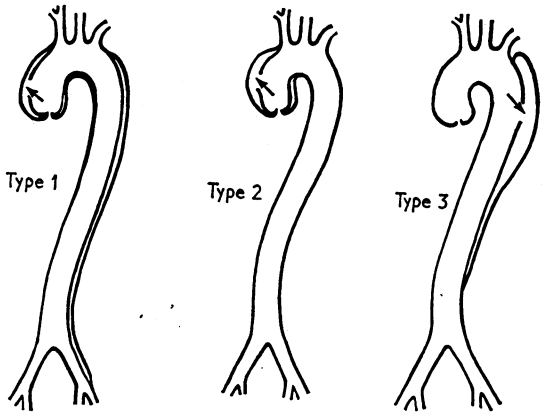

FIG. 1.—Classification of dissecting aorta aneurysms (De Bakey *et al.*, 1965).

Type 1 starts from a tear usually within a few centimetres of the aortic valve and extends distally through the thoracic and abdominal aorta and may reach the femoral vessels. The circumferential involvement is variable, the dissection often proceeding spirally. Proximal dissection occurs also to a variable extent. This may be associated with dislocation of the aortic annulus resulting in aortic regurgitation.

Type 2 is confined to the ascending aorta and carries the same risk of aortic regurgitation.

Type 3 starts in the distal aorta at the isthmus and progresses mainly distally. This is the most benign variety.

Natural History

The poor outlook for patients with dissecting aneurysms was well illustrated by Hirst *et al.* (1958), who reviewed 505 patients. Survival time was determined in 425 (Table I). A review of the experience at St. Mary's Hospital in the years 1962 to 1965 showed that with either conservative or surgical management the overall survival rate was not acceptable, there being only four survivors out of 32 patients admitted (Table II). Personal experience has convinced us that early surgical repair is extremely difficult and hazardous, and a programme was therefore designed to carry the patient through the acute stage by active hypotensive therapy. Subsequently hypotensive therapy could be continued on a long-term basis if serious coincidental disease precluded major surgery.

Altogether 24 patients have been treated in this way since 1965. Seven have been managed surgically with success and six are receiving continued hypotensive therapy. Eleven have died.

TABLE I.—*Mortality Rate in 425 Cases of Dissecting Aneurysm (from Hirst et al., 1958)*

Within 24 hours	3%
After 24 hours	21%
After 1 week	62%
After 2 weeks	74%
At 1 year	93%

TABLE II.—*Dissecting Aneurysms, St. Mary's Hospital* (1962–5)

Total	32
Surgical treatment (upper 1, lower 14)	15
Survivors	2
Medical treatment	17
Survivors	2

Clinical Features

The onset is usually abrupt, with anterior chest pain of great severity. The pain is often worse and more prolonged than that experienced in cardiac infarction, and may be described as of a tearing quality. Radiation of the pain through to the back is common, and downward spread can extend to the abdomen and even to the hips and legs. The patient presents a shocked appearance, though he may be normotensive or only mildly hypotensive. Pulse deficiencies are common but may be transient.

In type 1 lesions the pain is felt centrally in the chest with radiation to the back and abdomen. Carotid occlusion may cause neurological signs, often with variations of level of consciousness, while subclavian involvement causes pressure differences between the arms. An unusual manifestation is shown in Fig. 2: the left radial pulse rate is double the heart rate, owing to left subclavian filling from the true lesion and delayed secondary filling from the outer false lumen.

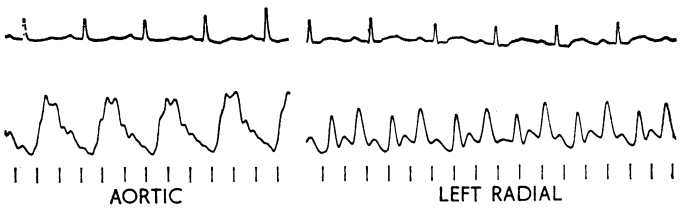

FIG. 2.—Double pulse wave in radial artery compared with aortic pulse. A physical sign of extension of a dissection into the subclavian artery.

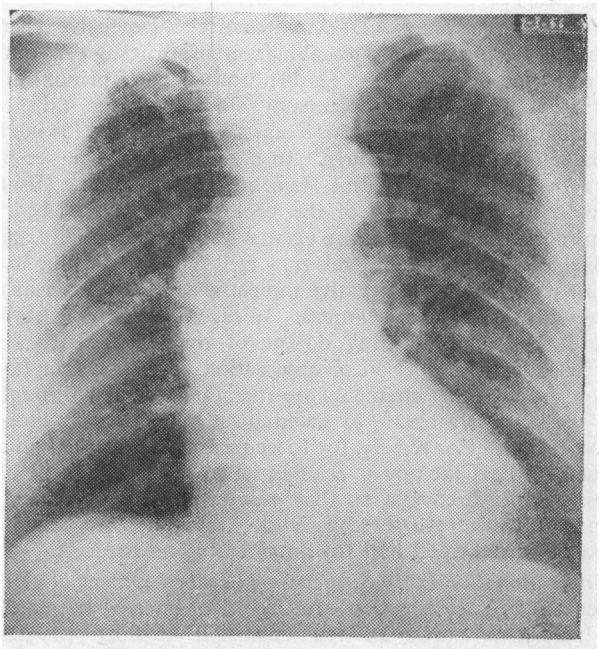

FIG. 3.—Widened mediastinal shadow in a type 1 aortic dissection due to dilatation of the ascending aorta.

Clinically, a type 1 dissection is strongly suggested by the appearance of an atrial dysrhythmia, aortic regurgitation, a pericardial rub, or pericardial electrocardiographic change. The serum aspartate aminotransferase is usually raised, so is of little value in differentiation from cardiac infarction.

In type 3 dissections pain is more commonly epigastric, with radiation to the legs, chest, or back. Pulse differences were noted in 7 of the 11 patients. Dysrhythmias and aortic regurgitation were not observed and pericardial changes were not seen on the electrocardiogram. Despite dependence on portable films, radiological signs of value include a widening of the mediastinal shadow (Fig. 3), especially in type 1 dissections, and a left pleural reaction of varying degree in both types. The film must be taken in full inspiration for the diagnostic widening to be conclusive, as in normal subjects a supine film in expiration shows a broad mediastinum.

Primary Management

We have generally followed the regimen suggested by Wheat *et al.* (1965). One considerable advantage of this programme is that it can be applied at any hospital, avoiding the need for moving an ill patient over long distances to a cardiothoracic centre. Primary treatment consists in relieving pain with diamorphine and administering oxygen if there is any respiratory distress. Frequent observations of blood pressure, level of consciousness, and peripheral pulses are recorded from the outset. A portable chest *x*-ray film and electrocardiogram are also taken as soon as possible.

Complete bed rest is mandatory. Hypotensive therapy is started when the patient is recovering from the acute shocked state. Care has to be exercised in the timing and vigour of the therapy applied, but, in general, an attempt is made to maintain the systolic blood pressure level at between 100 and 140 mm. Hg provided this level is sufficient to maintain consciousness and produce a good urine output.

Initial Hypotensive Therapy.—Reserpine 1–2·5 mg. by intramuscular injection six-hourly can be started in this acute phase and used for one to three days. Methyldopa (Aldomet) 250 mg. six-hourly is started as soon as oral therapy is practicable. In the occasional refractory case trimethaphan (Arfonad) 1–2 mg./ml. may be used by intravenous drip to control the pressure. In practice this has rarely been required.

Subsequent Treatment.—Maintenance hypotensive therapy is usually achieved by a combination of drugs given orally: (1) reserpine 0·1 mg. t.d.s., (2) bendrofluazide (Aprinox) 5 mg. daily, (3) potassium supplements (slow K 0·5 g. t.d.s.), (4) amylobarbitone (Amytal) 15–45 mg. six-hourly, and (5) methyldopa 250–750 mg. six-hourly. In patients who do not respond to methyldopa, guanethidine (Ismelin) or bethanidine (Esbatal) may be used. In view of the prolonged period of bed rest required, adrenergic blocking agents are not the initial drugs of choice. Propranolol (Inderal) may well replace reserpine in this regimen in view of its negative inotropic action, but our experience with this has been limited. In several patients it has been found that a diminishing dosage of hypotensive drugs was needed during the period of observation. Careful four-hourly blood pressure measurements are therefore essential throughout the weeks of treatment to avoid overtreatment and excessive hypotension. Conversely, for those patients discharged on continued medical therapy it is essential to ensure frequent blood pressure measurements as they resume normal activities and return to work. In these patients the hypotensive drugs may need to be increased and the amylobarbitone reduced.

Indications for Early Surgery.—The development of signs of extension of the dissection—for example, internal haemorrhage or persistent aortic branch occlusion causing progressive ischaemia of the brain or a limb—indicates the need to consider early surgery. This step was not taken in any of the patients of the series, but a fenestration (re-entry) procedure would still be the operation of choice in such circumstances (Rob and Kenyon, 1960).

Aortography.—Six to eight weeks after the onset an arch aortogram was performed by the femoral or axillary route. The objects were to confirm the diagnosis and to demonstrate the site of origin and extent of the dissection. The typical aortographic change includes the demonstration of a double channel in the aorta. The true lumen is deformed, showing a "twisted ribbon" appearance (Fig. 4). This is continued to the lower end of the dissection, and the abdominal aorta often appears tortuous. The false channel is usually ill-defined, as there is little flow through it unless re-entry has occurred spontaneously lower down.

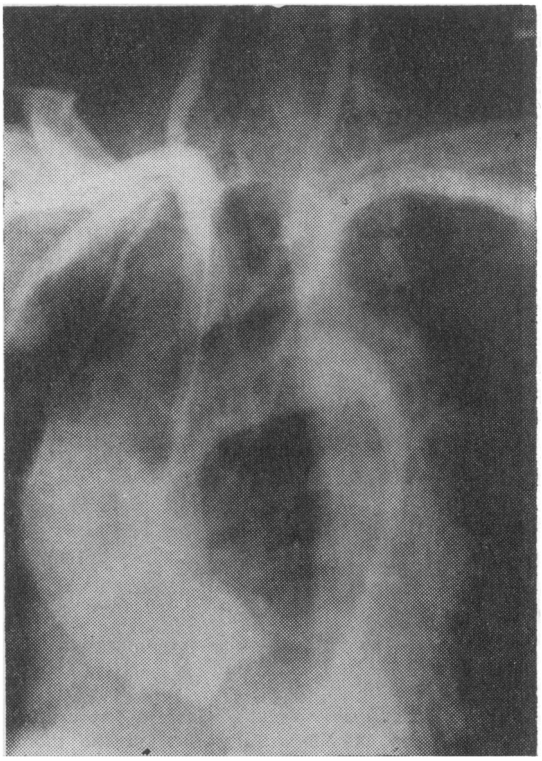

FIG. 4.—Aortogram in a type 1 dissection showing deformity of the ascending aorta and arch, narrowing of the descending aorta, and a "twisted ribbon" appearance.

Further Management

After aortography the patient can be assessed for further treatment. Patients selected for continued medical management have been those with concurrent disorders that contraindicated surgery, such as obesity, senility, and heart block.

Elective Surgical Reconstruction.—Surgery has been performed on nine patients. The indications have included increasing aortic regurgitation and severe claudication. The object of surgery has been to repair the intimal split and to restore blood flow through a single channel. Total cardiopulmonary bypass with coronary perfusion or mycardial protection by cooling is necessary for type 1 and type 2 dissections; partial left heart bypass only for type 3. In each type the aorta is transected 2–3 cm. beyond the apparent proximal end of the dissection as shown by aortography. This is usually close to the site of the intimal tear, which can be sutured direct to the serosal layer. The false channel is larger than the true lumen, and some tailoring is necessary before the aorta and intima can be resutured to form one layer. When this has

been completed on both sides of the aortic transection the aorta is reconstituted either by direct anastomosis or by the interposition of a Dacron graft. Reconstruction or replacement of the aortic valve is performed if necessary.

Results

The results of treatment of 24 patients are given in Table III. Five died within 48 hours of the onset of the dissection. One other died of massive pulmonary embolism at six weeks during hypotensive therapy. Aortography was attempted on 16 of the 18 survivors and was successful in 14, confirming the diagnosis and outlining the anatomy of the lesion.

Nine patients were selected for reconstructive surgery; all had undergone successful aortography. Four were type 1, and five suffered from type 3 dissections. One of each type died during or soon after surgery. The remainder recovered from the operation and have returned to their previous activities on oral hypotensive therapy. There have been no late deaths. The postoperative survival period extends from seven months to three and a half years.

TABLE III.—*Results of Treatment* (1965–8)

Total No. of cases	24
Deaths within 7 days	5
Patients surviving initial hypotensive treatment	18
Patients treated surgically (5 type 1, 4 type 3)	9
Operative deaths	2
Patients on prolonged hypotensive therapy	9
Deaths on hypotensive therapy	4
Survivors	13

Nine patients have been treated by continued hypotensive therapy alone. Three died within eight months of the initial onset, two of them from haemorrhage into the left chest. The others are alive and well within the limits of the coincidental disease which made them unsuitable candidates for surgery.

Discussion

Hypertension is one of the aetiological factors in dissecting aneurysms, as Hirst *et al.* (1958) showed in an extensive analysis of 505 cases. In a series of 290 deaths of known hypertensive patients (A. Breckenridge and C. T. Dollery, personal communication, 1968) there were 12 cases of acute dissecting aneurysm, an incidence of 4%. Nine of these were type 1 dissections, and haemopericardium was the usual cause of death. These figures reinforce our belief that type 1 dissection is the most lethal of the types. Moreover, the similarity of the terminal syndrome to that of acute cardiac infarction may cause it to pass unrecognized and hence unreported in the absence of a necropsy. Thus the true incidence of dissection may well be greater than the current figures suggest.

The medical management advocated by Wheat *et al.* (1965) was based on the contention that hypotensive treatment exerted two beneficial effects: lowering the systolic pressure and also the force of isometric ventricular contraction, thus reducing the tendency for extension of the dissection or rupture of the aneurysm. Other authors have had encouraging results with similar methods (Austen *et al.*, 1967; Harris *et al.*, 1967).

Our previous experience of surgery of the aorta in the first few days after onset of a dissection convinced us that the technical difficulties of early reconstruction are considerable. The aortic wall at this time is thin and oedematous and fails to hold sutures. This friability was not present in the aorta of our patients who survived the acute phase and came to surgery later. The outer layer of the dissection had become thickened and fibrosed and was much more suitable for reconstruction.

Thus a programme was designed to carry patients with acute dissections through to the healing stage by medical means, the diagnosis being made on the clinical history and physical signs, and confirmed initially by a chest *x*-ray examination. Conclusive diagnosis may be obtained by arch aortography, which defines the extent of the process, but this procedure is disturbing to the seriously ill patient, and the results in these circumstances are often equivocal. We believe the optimum time for aortography is between six and eight weeks after onset.

At this time the decision to perform reconstructive surgery is taken, bearing in mind that total cardiopulmonary or partial left heart bypass is required. The exclusion of patients with coincidental disease has undoubtedly influenced the results of surgery in our series, but we feel that this approach is justified at present. Subsequent experience may show that patients with type 3 dissections whose blood pressure is well controlled may continue to do well on a medical regimen. Only a larger series and longer experience will show whether the reconstructed aorta is less likely to late rupture than the aorta with a persistent dissection. Type 1 patients, however, would seem to be at a greater risk, and elective surgery may be more frequently indicated.

There appears to be a place for both medical and combined medical and surgical management in the treatment of the condition, and each case must be treated on its merits. It is a major advantage of the initial period of medical care that the patient can be treated in a local hospital through the acute phase of the illness. Subsequent transfer to a cardiothoracic units when the patient has recovered carries a smaller risk.

In reporting the early results of this approach it is realized that the numbers are small but the method appears encouraging. The overall survival in this small series is an improvement on most other series. The condition is sufficiently rare to prevent any one centre obtaining a very large experience, but it is hoped that others will adopt a similar programme of management to allow a more critical assessment to be made from a larger number.

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