ELSEVIER

European Journal of Cardio-thoracic Surgery 14 (1998) 449–452

EUROPEAN JOURNAL OF CARDIO-THORACIC SURGERY

# Acute ascending aortic dissection complicating open heart surgery: [1]



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Received 30 November 1997; revised version received 5 August 1998; accepted 11 August 1998

**Abstract**

**Objective**: This retrospective study was designed to assess the risks of acute ascending aorta dissection (AAD) as a rare but potentially fatal complication of open heart surgery. **Method**: Among 8624 cardiac surgical procedures under cardiopulmonary bypass (CPB) and cardioplegic myocardial protection from 1978 to 1997, 10 patients (0.12%) presented with a secondary or so called 'iatrogenic' AAD. There were seven men and three women, mean age 64 ± 9 years, ranging from 47 to 79. The original procedures involved five coronary artery bypass grafts (CABG), one repeat CABG, one aortic valve replacement (AVR), one AVR and CABG, one mitral valvuloplasty (MVP) and CABG and one ascending aorta replacement. We retrospectively analyzed their hospital records. **Results**: Group I consisted of seven patients with AAD intraoperatively and group II consisted of three patients who developed acute AAD 8–32 days after cardiac surgery. In group I, treatment consisted of the original procedure, plus grafting of the ascending aorta in six patients and closed plication and aortic wrapping in one. In group II, two patients received a dacron graft and one patient developed lethal tamponnade due to aortic rupture before surgery. Postoperatively, six patients responded well and three died (33%), two patients from group I on the 2nd post-operative day with severe post-anoxic encephalopathy, and one from group II with severe peroperative cardiogenic shock. **Conclusion**: Preventing AAD with the appropriate means remains standard practice in cardiac surgery. If AAD occurs, it requires prompt diagnosis and interposition graft to allow a better prognosis. Intraoperative AAD happens at the beginning of CPB jeopardizing perfusion of the supra-aortic arteries. 

*Keywords:* Aortic dissection; Cardiopulmonary bypass; Surgical complication; Surgical treatment

## 1. Introduction

Canulating the ascending aorta for cardiopulmonary bypass was first proposed by Nunez and Bailey in 1959 [1]. Their technique became widely accepted only in the late 1960's. Retrograde aortic dissection, a frequent mortal complication of cardiac surgery, was well known with reports as high as 3% in femoral canulation or secondary to aortic crossclamping [2]. In 1974, Williams et al. [3] and Reinke et al. [4] reported the first successfully treated acute aortoiliac dissections resulting from aortic canulation. Since then, a few papers [5–8] have reported this rare but potentially fatal complication, stating the importance of early diagnosis and prompt surgical repair. Current knowledge is based on conclusions of 'ancient' publications from the pre-cardioplegic era and a few recent reports [9–11]. We reviewed our own cases since the introduction of cardioplegic myocardial protection and analysed the early and long-term results.

## 2. Method and patients

Among 8624 cardiac surgical procedures under cardiopulmonary bypass (CPB) and cardioplegic myocardial protection from January 1978 to March 1997, we found 10 patients (0.12%) having presented a secondary or so called

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[1] Presented at the 11th Annual Meeting of the European Association for Cardio-thoracic Surgery, Copenhagen, Denmark, September 28 – October 1, 1997.

1010-7940/98/$19.00 
*PII* S1010-7940(98)00224-3

'iatrogenic' acute ascending aorta dissection (AAD). The retrospective review of their hospital records is the basis of this report. Follow-up information on surviving patients was obtained from direct patients' interview. If death had occured, the cause was established by contacting referring physicians or the Swiss federal registry of deaths. The study cases are divided into two groups based on the time of presentation of AAD. Preoperative data are summarized in Table 1.

Group I consisted of four men and three women (mean age 66 ± 7 years, ranging from 56 to 79) who developed AAD intraoperatively. Four patients were diagnosed with coronary artery disease, one with CAD and aortic stenosis, one with CAD and myxoid mitral regurgitation, and one with aneurysm of the ascending aorta.

Group II consisted of three men (mean age 58 ± 10 years, ranging from 47 to 68) where AAD appeared between the 8th and 32nd post-operative day (POD). Two patients were diagnosed with CAD and one with aortic regurgitation.

Eight patients had a history of hypertension, a well-known predisposing factor for dissection, who was directly responsible for dissection in two patients from group II.

The aortic diameter was clearly dilated in patient no. 7 at 58 mm with a thin wall. Patients nos. 6 and 9 were described as normal. The other patients had no conspicuous features.

Original operative procedures included coronary artery bypass grafting (CABG) in six, AVR and CABG in one, mitral valvuloplasty and CABG in one, aortic valve replacement (AVR) in one, and ascending aorta replacement in one.

### *2.1. Canulation and surgical technique*

Canulation technique was fairly homogeneous throughout the years. It consisted in double concentric subadventitial purse-string 4–0 sutures; sufficient adventitia is excised in their centre; adequate intimo-medial incision is made with a No. 11 bistoury blade. Predilatation with a conical curved dilator is performed before gentle insertion of a curved-tip non-wire reinforced 8 mm Sarns' aortic canula. Arterial pressure is systematically lowered before cannulating the aorta. Aorta was always cross-clamped with the Fogarty hydrogrip aortic clamp. Myocardial protection was established by anterograde intermittent cristalloid Saint-Thomas' cardioplegia.

## 3. Surgical results

Intraoperative findings are summarized in Table 2.

### *3.1. Group I*

Acute AAD occured intraoperatively in seven patients, the diagnosis was made after the start of the cardiopulmonary bypass in six cases. The intimal tear was localised at the canulation site in all six patients. Surgical treatment consisted of the primary procedure for all patients and open aortic repair with Dacron graft in five, failed closed repair followed by open aortic Dacron graft in one, closed plication and wrapping of the ascending aorta in one patient. Circulatory arrest in deep hypothermia to secure the intimal flap was necessary in three patients and to insert femoral canula in one.

No cardiogenic shock, no low output syndrome nor perioperative myocardial infarct occured in the first group. In two patients, despite an uneventful recovery from operation, we diagnosed a severe post-anoxic encephalopathy with classical EEG and CT scan pattern. Death occurred on POD 2 after medical support was stopped. In one patient impaired brain perfusion was suspected peroperatively due to a sudden drop in blood pressure at the left radial catheter while initiating bypass. In the other one there was absence of head cooling while installing general hypothermia. Autopsy revealed extended brain necrosis without carotid dissection.

Table 1
Patients preoperative data

| No. | Age | Sex | Diagnosis | Primary | AAD time | Hypertension | Aortic wall/diameter |
|---|---|---|---|---|---|---|---|
| Group I | | | | | | | |
| 1 | 62 | M | CAD | CABG | perop | 0 | NA |
| 2 | 62 | M | CAD | CABG | perop | + | NA |
| 3 | 68 | M | CAD | CABG | perop | ++ | (36*) |
| 4 | 71 | M | CAD + MR | CABG + MVP | perop | 0 | (32*)/thin |
| 5 | 79 | F | CAD + AS | CABG + AVR | perop | ++ | (32*)/thin |
| 6 | 56 | F | CAD | RedoCABG | perop | +++ | normal (20*)/thin |
| 7 | 64 | F | AsAA | As.AoR | perop | +++ | 58 mm (24*)/thin |
| Group II | | | | | | | |
| 8 | 47 | M | CAD | CABG | POD 9 | ++ | NA |
| 9 | 68 | M | CAD | CABG | POD 8 | +++ | normal (26*) |
| 10 | 58 | M | AR | AVR | POD 32 | + | NA |

M, male; F, female; CAD, coronary artery disease; MR, mitral regurgitation; AS, aortic stenosis; AsAA, ascending aorta aneurysm; AR, aortic regurgitation; CABG, coronary artery bypass grafting; MVP, mitral valvuloplasty; AVR, aortic valve replacement; AsAoR, ascending aorta replacement; POD, post-operative day; NA, not available, (*) diameter of the graft in mm.

Table 2
Peroperative data and follow-up

| No. | Site of tear | Aortic procedure/circulatory arrest | Aortic wall pathology | Surgical results | Long-term follow-up |
|---|---|---|---|---|---|
| Group I | | | | | |
| 1 | Canulation | Dacron graft (no) | NA | Discharged POD 16 | Died 79 m |
| 2 | Canulation | Suture + Wrapping (no) | ATS | Discharged POD 12 | Died 8 m |
| 3 | Canulation | Dacron graft (25′) | ATS | Brain death POD 2 | – |
| 4 | Canulation | Dacron graft (25′) | ATS | Brain death POD 2 - | – |
| 5 | Canulation | Dacron graft (40′) | CMN | Discharged POD 21 | Alive 71 m |
| 6 | Canulation | Dacron graft (6′) | CMN | Discharged POD 10 | Alive 89 m |
| 7 | Canulation | Dacron graft (no) | CMN | Discharged POD 9 | Alive 16 m |
| Group II | | | | | |
| 8 | SV anastomosis | Dacron graft (no) | Normal | Died on CPB | – |
| 9 | SV anastomosis | Dacron graft (no) | Normal | Discharged POD 8 | Alive 61 m |
| 10 | Aortotomy | Nihil | CMN | Died before surgery | – |

m, month; SV, saphenous vein; ATS, atherosclerosis; CMN, cystic media necrosis; POD, postoperative day.

### *3.2. Group II*

Acute AAD happened 8th, 9th and 32nd day postoperatively, secondary to a hypertensive crisis in two patients. One patient died of cardiac tamponnade on ascending aorta rupture before surgical repair, an autopsy was performed. One patient did well after aortic graft with reimplantation of the saphenous vein. One died under CPB because of intractable cardiogenic shock.

### *3.3. Pathology*

Histological examination of the aortic wall showed the presence of four cystic medial necrosis, three atherosclerosis and two described as normal.

### *3.4. Late results*

Follow-up was complete in six hospital survivors. At a mean time of 54 ± 33 (range 8–89) months, four patients were doing well and two are dead. The one patient with a closed plication presented an aortic rupture with cardiac tamponnade 8 months later. The second one suffered a recurrent myocardial infarction with ventricular fibrillation 79 months postoperatively.

## 4. Discussion

To avoid vascular complication associated with cardiopulmonary bypass we adopted, like each cardiac surgeon, preventive measures. Most injuries described in the literature were related to aortic canulation [3], aortic crossclamping [6], partial lateral occlusion [7], surgical intimal disruption like aortotomy and vein anastomosis [8]. Like in the study of Taylor et al. [12], all dissections in our group I were due to canulation. Dissections in group II involved surgical intimal disruption associated with hypertension.

Intra-operative recognition of aortic dissection requires a high index of suspicion and good clinical judgment. Diagnosis was made on appearance of a tense, circumferential dilatation with bluish discoloration of the exposed aorta, enlarging during CPB via the aortic canula.

Cross-sectional trans-esophageal echocardiography (TEE) introduced by Hanrath opened up a new tool to detect aortic dissection (see [13]). We routinely use it for every cardiac procedure since 1992. TEE is a valuable intraoperative tool like direct aortic peroperative echography to differentiate between dissection and subadventitial hematoma. The two most recent patients underwent these examinations, identifying the dissection and evaluating permeability and perfusion of the supra-aortic arteries.

As with acute dissections, generally, this complication carries a mortality of 90% when untreated. The most frequent cause of death is full thickness rupture with cardiac tamponade. This sudden death was encountered in patient no. 10 at the 32nd postoperative day before surgery could intervene.

Murphy et al. [14] attributed their 33% mortality rate to subsequent myocardial dysfunction due to prolongation of crossclamping time. Based on our experience in nine operated patients, only one died from myocardial dysfunction. We think that since introduction of routine myocardial protection, duration of crossclamping time might play a less important role in early mortality. Interposition graft has to be therefore performed.

Ohashi et al. recently reported cerebral malperfusion as a consequence of iatrogenic dissection [11]. Two patients in our series showed the same complication. Autopsy revealed no evidence of carotid dissection so the mechanism of malperfusion is likely to be blood flow in the false lumen with total occlusion of supra aortic trunks.

Morphological studies have shown that cystic medial

necrosis, typical of connective tissue disorders, is only quantitatively different from the histopathologic features found in the normal aging aorta [15]. These features represent the morphologic expression of an interaction within the aortic wall of traumatizing and reparative processes due to hemodynamic impact. The high incidence of cystic media necrosis found in our histological studies of aortic wall is directly due to the high incidence of hypertension and the elevated mean age of our collective.

## 5. Conclusion

Preventing AAD with the appropriate means remains standard practice in Cardiac Surgery. If AAD occurs, it requires prompt diagnosis and interposition graft to allow a better prognosis. Intraoperative AAD reveals itself at the start of CPB jeopardizing perfusion of the supra-aortic arteries. We recommend to check routinely canulation site before starting CPB. If dissection is suspected, TEE will confirm if it is necessary to change the site of arterial canulation.

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