Traumatic Laceration of the Aorta

Rapid deceleration, which occurs in high-velocity automobile accidents, forces passengers against the steering wheel or dashboard and produces a crushing injury to the bony thorax. Rapid deceleration also sometimes tears the thoracic aorta. This tearing usually occurs where the movable aortic arch becomes fixed to the posterior chest wall, namely, at the level of the left subclavian artery; however, such tears have been noticed elsewhere in the thoracic aorta, principally in the ascending aorta at the level of the innominate artery and in the transverse arch itself. The actual incidence of such injuries is unknown, but it is clear that they are related to the actual incidence of automobile accidents producing rapid deceleration and, therefore, are probably increasing in frequency. Aortic tears, even when circumferential, may produce hemorrhage which will be contained by the intact adventitia of the aorta in the form of a periaortic hematoma. It is now established that the natural history of these hematomas is eventual rupture into the pleural cavity, but, indeed, they may be contained for a long time and actually have been reported as existing in a contained fashion years after the occurrence of injuries.

It is essential that all patients who sustain injuries due to rapid deceleration be evaluated immediately for the possibility of aortic laceration. The hematoma is usually apparent on a plain x-ray film of the chest, where it produces a widening of the mediastinal shadow. Compression of the descending thoracic aorta can ordinarily be recognized because it produces a hypertension in the brachiocephalic circulation and a diminution in pulse pressure in the lower extremities. The recognition of any of these signs indicates that emergency aortographic studies should be performed to identify the site of laceration. Aortographic demonstration of a laceration should be followed by an immediate emergency operation to repair the laceration. Only active bleeding at another site takes precedence.

A variety of techniques have now been developed which permit the descending thoracic aorta to be isolated from the remainder of the aortic circulation, and thereby resected or repaired. These include the establishment of a left atrial-femoral bypass; the establishment of a femoral-femoral bypass employing a pump oxygenator; the use of temporary shunts which are relatively nonthrombogenic and permit bypass of the aorta without the use of systemic administration of heparin; and, finally, rapid repair of the aorta with cross-clamping above and below the site of injury without the aid of any bypassing technique. The use of coated shunts, which can easily be inserted into the ascending aorta and the femoral artery prior to disturbing the hematoma, is a preferred technique, since it eliminates the systemic administration of heparin in the face of multiple injuries. It has recently been demonstrated that such shunts can be inserted between the apex of the left ventricle and the femoral artery; however, if there is active bleeding into the pleural space at the time of thoracotomy, or indeed, if the shunt cannot be placed without disturbing the hematoma, rapid cross-clamping of the aorta above and below, followed by evacuation of the hematoma and repair of the aorta by simple suture or by the interposition of a prosthetic graft segment can be performed. A report by Fry and associates in the July issue of Chest documented a case of traumatic laceration of the aorta which produced acute left ventricular failure, and which was managed by aortic resection and graft replacement using a left atrial-femoral bypass. The appearance of acute left ventricular failure as a principal sign of a periaortic hematoma in the descending thoracic aorta is distinctly unusual. The degree of aortic narrowing at this site which is necessary to produce acute left ventricular failure is severe (probably in excess of 60 percent). Usually such hematomas are recognizable from conventional signs (such as mediastinal widening or a difference in the pressures above and below the site of aortic compression) before a significant degree of luminal narrowing occurs, which can produce left ventricular decompensation. In addition, acute left ventricular failure may be a consequence of cardiac contusion itself and not related to luminal narrowing at
the site of the laceration. The use of the term, "traumatic coarctation of the aorta," for aortic compression by a periaortic hematoma is probably confusing. Although it is true that proximal aortic compression producing luminal narrowing greater than 60 percent will produce an increase in impedance to aortic blood flow, which will result in acute left ventricular decompensation, and although it is also true that left ventricular failure frequently occurs in congenital coarctation of the aorta, the pathophysiology in this latter condition is very different indeed. Acute left ventricular decompensation complicating congenital coarctation of the aorta within the first few days or weeks of life correlates poorly with the degree of aortic obstruction at the site of coarctation and is almost certainly a consequence of this obstruction combined with other abnormalities, such as additional obstructions of the aortic valve or ascending aorta, excessive pulmonary blood flow from ventricular septal defects, underdevelopment and relative hypoplasia of the left ventricle, and ductal closure in those circumstances where the ductus has been acting as a vent for excessive pulmonary blood flow. Similarly, in chronic coarctation, left ventricular decompensation is a consequence of the hypertension which develops in longstanding coarctation. The pathogenesis of this hypertension is incompletely understood.

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REFERENCE


Deaths Associated with Flexible Bronchoscopy

Flexible fiberoptic bronchoscopy was first introduced in this country in 1968. Since then, a number of clinical studies have clearly documented its value in the evaluation and management of a variety of bronchopulmonary diseases.

One of the remarkable aspects of flexible fiberoptic bronchoscopy is the low morbidity associated with this procedure. In 1974, on the basis of the analysis of a questionnaire survey, Credle et al1 reported that the incidence of all complications associated with flexible fiberoptic bronchoscopic examination was less than 0.1 percent. In a recent issue of Chest, Suratt and his colleagues2 reported the results of a more recent survey in which the overall complication rate was 0.3 percent. Of greater significance, 12 deaths and 45 life-threatening complications are included in their report.

Although the true mortality cannot be accurately determined by analyzing the information obtained from the type of survey conducted by Suratt and his co-workers,2 it is clear that a fatality associated with the use of the flexible fiberoptic bronchoscope is an extremely rare occurrence. Indeed, most comparable diagnostic procedures have death rates higher than that of the flexible bronchoscopic technique. For example, the reported mortality for cardiac catheterization and for pulmonary angiographic studies is greater than 0.1 percent.3,4 Despite the relative safety of flexible fiberoptic bronchoscopic examination, it is important to carefully analyze the deaths and life-threatening complications associated with this procedure.

In the series of Suratt et al,2 two patients died while actively bleeding from endobronchial tumors that had been manipulated during the procedure. Life-threatening hemorrhage also occurred in 11 other instances in which brush or forceps biopsy of an endobronchial lesion was performed. The remaining ten deaths in their report were attributable to sudden cardiac arrests. Each of the five patients whose age was recorded was over 67 years of age. Coronary arterial disease or severe chronic obstructive airway disease was present in the majority of these patients. Two patients died after administration of local anesthetics but before bronchoscopic examination was performed. In four other patients, cardiac arrests occurred after hyperventilation or hypotension developed following the administration of general anesthesia or morphine. A patient who was "in extremis" at the time of the procedure, a 70-year-old patient with advanced obstructive airway disease, and a 94-year-old patient also died suddenly during the procedure.

Based on these data, it appears that cardiac arrhythmias associated with premedication or the administration of anesthetics are the major causes of death associated with flexible fiberoptic bronchoscopic examination and that elderly subjects who have severe obstructive pulmonary disease or coronary arterial disease comprise a high-risk group. The importance of adverse drug reactions is further emphasized by the fact that 34 of the 45 life-threatening complications reported in the survey of Suratt et al2 were probably caused by drug administration.

The critical message of the report of Suratt and associates2 is clear: precautions must be taken when flexible fiberoptic bronchoscopic examination is considered in elderly patients. First, the indications for the procedure in such patients must be critically con-