Large aneurysms of the ascending aorta occasionally cause severe hemodynamic disturbance by compression. We describe the case of an 18-yr-old man who presented with dyspnea and developed hemodynamic collapse during computed tomography scanning. Computed tomography during resuscitation revealed that the aneurysm was compressing both the right pulmonary artery and the left bronchus. Emergency surgery was performed, but the patient died because of generalized cerebral infarction.

In most cases of aortic aneurysm, acute hemodynamic collapse only occurs if the lesion ruptures or if aortic dissection develops. If an aneurysm remains unruptured while it enlarges, it may compress the pulmonary artery and parts of the tracheobronchial tree. However, it is rare for hemodynamic collapse to occur without rupture or dissection, even if the aneurysm is very large. We present the unusual case of a young patient who developed a large aneurysm of the ascending aorta that remained unruptured and compressed the right pulmonary artery and left mainstem bronchus, causing hemodynamic collapse.

Case Report

An 18-yr-old man with pectus excavatum was admitted to hospital complaining of shortness of breath. He had a breathing problem for approximately 1 yr, but it had become significantly worse in the 2 mo before his presentation. Electrocardiogram showed a normal sinus rhythm (rate, 92 bpm), his arterial blood pressure was 140/80 mm Hg, and his respiration rate was 28 breaths/min. Lung examination revealed mild wheezing over the left hemithorax. A chest radiograph showed dilation of the ascending aorta, and thoracic computed tomography (CT) was scheduled. After lying down on the CT table and just before contrast injection, the patient went first into severe hypoxia and bradycardia and then into asystolic cardiac arrest. His trachea was intubated immediately, and cardiopulmonary resuscitation (CPR) was performed by the hospital resuscitation team (including an anesthesiologist). Only the scanning of the upper half of the thorax had been completed before the patient suffered a cardiac arrest (Fig. 1). He was diagnosed with aortic aneurysm and was transferred to the intensive care unit under CPR. Assessments during CPR in the intensive care unit revealed severe hypoxia (\( \text{PaO}_2 \), 32 mm Hg; \( \text{PCO}_2 \), 58 mm Hg; and pH value of 7.26), hypotension (systolic blood pressure between 40 and 50 mm Hg), and bradycardia (rate between 20 and 30 bpm). None of these conditions responded to epinephrine 1 mg every 3 min, a total dose of 3 mg of atropine, and 100% oxygen. Methylprednisolone (1 g) was administered.

Emergency surgery was performed because hypoxia was severe and sustained (arterial blood gas \( \text{PaO}_2 < 25 \) mm Hg), and the left lung was not ventilating adequately despite proper CPR. Arterial blood gas analysis performed immediately before the operation revealed a \( \text{PaO}_2 \) of 24 mm Hg, a \( \text{PaCO}_2 \) of 68 mm Hg, and a pH value of 7.20. During the procedure, cardiopulmonary bypass (CPB) was established immediately via the femoral artery and vein. This led to rapid improvement in oxygenation. The anesthetic drugs administered at the beginning of CPB were midazolam (7 mg) and fentanyl (5 \( \mu \)g/kg). The total time between cardiopulmonary arrest and initiation of CPB was 35 min.

Flexible bronchoscopy performed during CPB showed compression of the left main bronchus, and intraoperative transesophageal echocardiography demonstrated a large aneurysm of the ascending aorta that measured 11 cm in diameter. Cooling was begun immediately to repair the aneurysm, and the patient’s chest was opened via a median sternotomy. Exposure of the pericardium revealed the huge aneurysm occupying a large portion of the pericardial space (Fig. 2). Thiopental (1 g) was administered for the protection of the central nervous system. The patient was cooled to
18°C, and total circulatory arrest was induced. Aortotomy showed that there was no aortic dissection, and selective antegrade cerebral perfusion was started immediately via cannulation of the innominate artery and the left common artery. The left subclavian artery was occluded using a Fogarty catheter. The distal aorta was transected immediately below the innominate artery, and a prosthetic graft was anastomosed to the distal portion of the aorta. Circulatory arrest with selective antegrade cerebral perfusion lasted 23 min. The proximal portion of the aortic graft was anastomosed to the supracoronary ascending aorta. The patient was weaned from CPB without any inotropic drugs.

Because large doses of atropine and epinephrine had been administered during CPR, it was not surprising that the patient’s pupils were dilated during the intraoperative and early postoperative periods. The patient never regained consciousness after surgery, therefore a cranial CT was performed 7 h after the surgery. The scan showed generalized infarction and edema throughout the brain. There was no activity on electroencephalography. Brain death was diagnosed via single-photon-emission CT, and the patient was transferred to another health care facility in accordance with the family’s wishes for transplantation. Pathological examination of the aortic aneurysm exhibited the classic features of cystic medial degeneration.

Discussion
Thoracic aneurysms only cause signs and symptoms if they compress adjacent structures or rupture. Large aneurysms of the ascending aorta can obstruct the superior vena cava or innominate vein and produce venous distention throughout the upper extremities and neck. Most aneurysms that obstruct the trachea or bronchi are located in the aortic arch. Occasionally, an aneurysm of the ascending aorta may be marked compression of the trachea and obstructive atelectasis of the upper lobe of the right lung (1). Pulmonary artery compression caused by aortic dissection is rare, but, in such cases, if the right pulmonary artery is compressed, the clinical signs may mimic pulmonary embolism (2,3). Sugimoto et al. (4) described one patient with a large aneurysm of the aortic arch that caused severe hypoxia by compressing the left main bronchus and the right pulmonary artery. Our patient had a similar form of compression but his lesion was an aneurysm of the ascending aorta.

The right pulmonary artery and the ascending aorta share common adventitia that fixes these vessels together. The right pulmonary artery is located between the ascending aorta and the vertebral column. Because of this fixation and vessel positioning, marked dilation of the ascending aorta can lead to pulmonary artery compression (5). Further, an abnormality such as pectus excavatum could prevent an aneurysm from expanding anteriorly. In this setting, enlargement of an aneurysm between the sternum and the vertebral column could indirectly compress the right pulmonary artery. In our case, the patient had pectus excavatum and the aneurysm expanded to the point where it compressed both the right pulmonary artery and the left main bronchus. The patient stated that his dyspnea had worsened with time. We suspect that continued enlargement of the aneurysm led to pulmonary artery compression and severe ventilation-perfusion mismatch, causing severe hypoxia. We believe that the
acute hemodynamic collapse during CT was probably brought on by aggravated compression of the right pulmonary artery caused by body positioning (supine position) and recent rapid enlargement of the aneurysm. These factors likely led to almost total occlusion of the right pulmonary artery and left mainstem bronchus by the aneurysm.

Stoppage of spontaneous ventilation and subsequent endotracheal intubation results in loss of negative pressure in the thorax, and this places positive pressure on the mediastinum. In a patient whose right pulmonary artery is being compressed by an aortic aneurysm, the above-mentioned positive pressure in the thorax can reduce systemic venous return, thus further compromising pulmonary circulation (6). The latter mechanism probably contributed to our patient’s lack of improvement in oxygenation during CPR, although this technique was performed correctly, and 100% oxygen was administered.

Our case emphasizes that hemodynamic collapse can occur in a patient with an aneurysm of the ascending aorta. It also demonstrates that this acute collapse can occur even in the absence of dissection or dissection-related complications, such as rupture, aortic valve regurgitation, cardiac tamponade, myocardial infarction, or acute hypovolemia. In our patient, the collapse resulted from compression of the right pulmonary artery and the left mainstem bronchus by a large aneurysm of the ascending aorta. CPR is inadequate because sternal compression during resuscitation may further increase severe hypoxia via the compressing effect of the aneurysm on the neighboring structures, such as the right pulmonary artery or left bronchus. Emergency CPB is a prompt alternative in these patients with cardiac arrest who do not respond to standard CPR. In this patient, the postresuscitation neurological signs and electroencephalogram patterns were not evaluated after initiating CPB. The neurological state of the patient could have been evaluated for predicting neurological outcome. However, it would still not be possible to predict the extent of anoxic brain damage precisely, even if this evaluation had been done. In these patients, emergency surgery with CPB support is the only potentially life-saving option if a short period of CPR is unsuccessful.

References