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Cardiopulmonary Bypass Complicated by Inadvertent Carotid Cannulation

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The use of the ascending aorta as the site of arterial cannulation during cardiopulmonary bypass was reported in 1957.[1] Since then, the ascending aorta has become the preferred site for arterial return during procedures utilizing the pump oxygenator for total cardiopulmonary bypass. The risks of inadvertent carotid cannulation and other complications of this technique have been recognized in reviews of complications of aortic cannulation.[2,3] Actual case reports of inadvertent carotid cannulation[4,5] have been limited to the surgical literature. Obviously, the anesthesiologist should be aware of and able to promptly recognize this complication. Here we report a case of inadvertent carotid cannulation.

REPORT OF A CASE

A 22-year-old man was admitted with a two-to-three-year history of lightheadedness and blurred peripheral vision during strenuous exercise. At the age of 2 years, a coarctation of the aorta had been repaired. His general health was otherwise good. Positive physical findings were limited to a grade IV/VI systolic murmur in the aortic area which radiated into the neck and diminished the left radial and brachial pulses. Cardiac catheterization demonstrated a systolic gradient of 76 torr across the aortic valve. Cineangiography showed slight left ventricular dilatation, symmetric left ventricular contraction, and a bicuspid, stenotic aortic valve without regurgitation. There was minimal narrowing of the aorta at the site of the previous coarctation. The left subclavian artery appeared to arise from the inferior portion of the narrowed segment.

The patient was prepared for surgical repair or replacement of the aortic valve. After local anesthesia of cannulation sites, two large peripheral intravenous cannulae, a central venous cannula, and a right radial arterial cannula were placed. Anesthesia was induced with thiopental and maintained with nitrous oxide and halothane. Pancuronium was used to provide muscle relaxation. During induction of anesthesia and median sternotomy, arterial blood pressures ranged from 100/55 torr to 140/80 torr and mean central venous pressures from 4 torr to 8 torr. The aorta was cannulated with a 24-French Sarns flexible cannula with a movable marker ring. Upon initiating total cardiopulmonary bypass (CPB), mean arterial pressure (MAP) was initially 30 torr. The administration of phenylephrine, 1 mg, caused MAP to rise briefly to 50 torr. Despite cross-clamping the ascending aorta and additional increments of phenylephrine, MAP remained at 40–45 torr. Pump flow was maintained at 2.2 $l \cdot m^{-2} \cdot min^{-1}$ or more, and venous return to the pump was good. After approximately 50 min of CPB, rhinorrhea and slight facial swelling in the vicinity of the left parotid gland were noticed. The persistent hypotension was accompanied by a urinary output of 20 ml despite the intravenous administration of furosemide, 20 mg, and mannitol, 12.5 g, during 68 min of CPB. Arterial blood-gas values shortly before completion of CPB were P_{O_2} 65 torr, P_{CO_2} 41 torr, and *p*H 7.28. Replacement of the aortic valve with a #9 Starr-Edwards prosthesis was completed. After CPB was discontinued, a systolic blood pressure of 70 torr was maintained while left ventricular pressure was 170–180 torr. Because of the high gradient across the prosthetic valve, CPB was reinstituted and a #23 Bjork-Shiley prosthetic valve was placed in the aortic position.

During the second period of CPB, MAP was initially 35 torr; it decreased to 25 torr with pump oxygenator flows in excess of 2.2 $l \cdot m^{-2} \cdot min^{-1}$. Urinary output was nil. The swelling near the left parotid gland increased, became indurated, and was accompanied by a profuse clear rhinorrhea, marked conjunctival edema, and left serosanguineous otorrhea. The aortic cannula was palpated and was found to be high in the aortic arch. When the cannula was withdrawn 4 cm, MAP promptly increased to 75 torr, and there was an apparent decrease in saturation of the venous blood returning to the oxygenator.

This sequence of events led to a diagnosis of inadvertent cannulation of the left carotid artery. This probably resulted from anatomic distortion of the aortic arch and origin of the left subclavian artery by the earlier resection of the coarctation.

After completion of the valve replacement, CPB was discontinued, using isoproterenol and calcium chloride to maintain adequate cardiac output. Epicardial pacing was employed to treat atrioventricular dissociation. Urinary output improved following CPB. Seventy-five minutes after bypass an episode of ventricular tachycardia progressed to ventricular fibrillation, but it did not recur after defibrillation and administration of intravenous lidocaine and procainamide.

Immediately following the surgical procedure the left pupil was dilated and slightly reactive to light, while the right pupil was dilated and nonreactive. Following antagonism of neuromuscular blockade, the patient was moving all extremities and was responding appropriately to command. An electroencephalogram at that time was diffusely and continuously abnormal, with lower voltages over the left hemisphere. Arterial blood-gas analysis revealed persistent metabolic acidosis.

Normal neurologic function returned by the first postoperative day. The trachea was extubated, and adequate arterial blood-gas values were maintained. Later that day, pacemaker failure, low cardiac output, and decreasing renal function developed. There were marked increases in serum creatine phosphokinase, lactate dehydrogenase, glutamic oxaloacetic transaminase, potassium, BUN, and creatinine. The trachea was reintubated, and the patient needed dopamine and norepinephrine for treatment of hypotension and low cardiac output. On the second postoperative

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day, the patient's neurologic status had deteriorated, abdominal distention developed, and he was successfully resuscitated from several cardiac arrests. Peritoneal dialysis was begun for acute renal failure. Slight hemodynamic improvement occurred on the third day after a tube cecostomy was accomplished using local anesthesia. Cardiac arrest occurred again on the fourth day, following which the patient was semicomatose but responsive to painful stimuli. On the fifth day, despite maximal supportive measures, he had another cardiac arrest, from which he was not resuscitated. Autopsy was not performed.

Discussion

Previous reports of inadvertent cannulation of a carotid artery as a complication of CPB are limited to those by Kulkarni[4] and Krous *et al.*[5] In each of these reported cases the patient failed to regain consciousness and had evidence of a severe cerebral injury. In Kulkarni's[4] patient, pressure in the arterial cannula rose to more than 300 torr at a flow rate of 3 l/min. In Krous'[5] patient, systemic MAP was 30–55 torr at a pump flow rate of 2.8 l/min. That patient also had left-sided facial edema, lacrimation, petechiae and serosanguineous otorrhea. At autopsy the left carotid artery was found to arise laterally from the aortic arch, which placed its ostium in the path of the perfusing jet from the aortic cannula.

These unfortunate cases have several common features, which may make avoidance or earlier recognition of this entity possible. 1) Abnormalities of the aortic arch, or the vessels arising from it, can favor cannulation of the carotid artery or other arch vessels. Positioning of the aortic cannula should be such that the tip is directed along the inferior aspect of the aortic arch during CPB. 2) Perfusion into a single vessel (*e.g.*, the carotid artery) instead of the proximal aorta would result in increased pressure in that artery and in the arterial tubing from the pump oxygenator, as reported by Kulkarni.[4] There would be a consequent decrease in systemic perfusion pressure measured elsewhere, as reported by Krous *et al.*,[5] and vasoactive drugs would not be expected to effectively raise the pressure measured in other systemic vessels. 3) When MAP during CPB is unexpectedly low and does not respond to vasopressors, unilateral otorrhea, facial edema, petechiae, conjunctival edema and rhinorrhea should be sought to confirm a suspected carotid cannulation. 4) Direct carotid cannulation would result in increased mean cerebral perfusion pressure,[6] and would be expected to elevate intracranial pressure, favor cerebral edema, and even disrupt capillaries within the brain.[4] Despite adequate venous drainage and an intact circle of Willis, overperfusion of the cerebral circulation should cause rapid changes in the electroencephalogram. 5) Other causes of hypotension during cardiopulmonary bypass must be considered, and would include: allergic reactions to drugs or blood, pharmacologic alpha blockade, obstruction of the arterial tubing or cannula, intramural (medial) cannulation of the aorta, or aortic regurgitation in the presence of a ventricular vent.

We believe that the patient who has sustained carotid cannulation during CPB must be considered to have suffered a severe cerebral injury, and that aggressive therapy to monitor and treat cerebral edema and increased intracranial pressure must be instituted. While cerebral injury in such a patient may be potentially reversible if detected early, repeated episodes of marginal or reduced cerebral perfusion can be anticipated to be poorly tolerated.

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