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Hypoxemia Following Cardiopulmonary Bypass

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Pulmonary dysfunction characterized by widening of the alveolar–arterial oxygen gradient (A-aD_{O_2}), common following cardiac surgery,[1] is usually secondary to preexisting pulmonary disease, intraoperative pulmonary damage,[2] or postoperative pulmonary edema.[3] We present two cases of severe hypoxemia following cardiopulmonary bypass (CPB) in which therapeutic maneuvers to improve the A-aD_{O_2} aggravated the hypoxemia. Further investigations demonstrated a right-to-left intracardiac shunt through a patent foramen ovale that had been physiologically closed preoperatively.

We wish to emphasize this recognized complication after CPB, the importance of recording a patent foramen ovale at the preoperative heart catheterization, and the opportunity for surgical correction. The value of clinically recognizing this cause of postoperative hypoxemia is emphasized by the rapid response to appropriate therapy. The diagnosis can be made at the bedside using dye-dilution curves[4] and by analysis of arterial blood gases, as demonstrated by the following cases.

REPORT OF TWO CASES

Patient 1. A 52-year-old man who had incapacitating angina was admitted for elective aortocoronary bypass grafting. Cardiac catheterization revealed triple-vessel coronary disease with a probe-patent foramen ovale. The pressure gradient between the atria was normal [peak left atrial pressure (P_{LA}) 15 torr; peak right atrial pressure (P_{RA}) 10 torr]. At preoperative evaluation, the roentgenogram of the chest and arterial blood-gas values while the patient was breathing room air were normal. He underwent aortocoronary bypass, with 158 minutes of extracorporeal circulation. After operation and transfer to the intensive care unit, ventilation was controlled, with V_t = 750 ml, f = 12/min, F_{IO_2} = 0.90. Initial analysis of arterial blood gases showed an alarming alveolar–arterial oxygen gradient (>600 torr), as the Pa_{O_2} was 55 torr (temperature corrected), with Pa_{CO_2} 45 torr and *p*H 7.38. Peripheral perfusion when assessed by clinical examination, and urinary output was adequate. The chest roentgenogram was completely clear. The left atrial monitoring line was in the left pulmonary vein. The addition of 5 cm H_2O positive end-expiratory pressure (PEEP) reduced Pa_{O_2} to 42 torr. Blood samples taken simultaneously from the pulmonary vein (with F_{IO_2} 0.9) via the left atrial line had a P_{O_2} of 443 torr. The presence of an atrial septal defect with a right-to-left shunt causing hypoxemia was suspected. Right atrial pressures (CVP = 16 torr) were found to be greater than pulmonary venous pressure (12 torr) at the time. An echocardiographic contrast study was unsatisfactory, with inadequate opacification of right or left heart. To reduce right ventricular afterload, intermittent mandatory ventilation (IMV) at a rate of 4/min with zero end-expiratory pressure was instituted. The Pa_{O_2} increased to 63 torr (F_{IO_2} = 0.80), and pulmonary venous blood P_{O_2} decreased to 275 torr. To make a definitive diagnosis, cardiac catheterization was performed. The Cournand catheter inserted via the femoral vein passed readily through an interatrial communication to the left atrium. At this time no difference between peak P_{LA} and peak P_{RA} was found (16 torr).[5] An increase in the right atrial blood oxygen saturation near the "defect" was found. There was a step-up in the blood oxygen saturation at the level of the mid-right atrium (85 per cent) compared with the inferior vena cava (72 per cent) and the low right atrial sample 76 per cent). The calculated shunt fraction from the data collected at this time was approximately 50 per cent. The dye-dilution curve showed an early-appearance hump and a slow "descent" (continuous recirculation type).[4] With these data we felt a bidirectional shunt across the interatrial opening should be considered.[5] Right atrial injection of contrast medium showed this shunt through an atrial septal communication.

The roentgenogram of the chest remained normal, and the patient was weaned rapidly to low F_{IO_2} levels in spite of persisting hypoxemia. The trachea was extubated in spite of persisting hypoxemia [Pa_{O_2} = 54 torr delivered by T piece (F_{IO_2} = 0.50)], and recovery was uncomplicated. After extubation Pa_{O_2} rose to 62 torr, with supplemental oxygen, 3 l/min, delivered with nasal prongs, and CVP decreased to 10 torr.

Anticoagulation was done to prevent paradoxical systemic embolization during the recovery period. The patient was discharged from hospital without further complication.

Patient 2. A 50-year-old man with coronary-artery disease and severe aortic insufficiency underwent aortic valve replacement and single coronary-artery bypass grafting. With F_{IO_2} 0.21, the Pa_{O_2} was 66 torr preoperatively. The operative course was unremarkable. The immediate post-cardiopulmonary bypass blood-gas analysis in the intensive care unit showed a marked A-aD_{O_2} (Pa_{O_2} 66 torr) with controlled ventilation (V_t = 850, f = 12, F_{IO_2} = 0.9). Roentgenogram of the chest showed no pulmonary infiltrate to account for this gradient; however, the left atrial monitoring line was found to be in the left pulmonary vein.

Simultaneous sampling from these lines revealed a large gradient (arterial blood P_{O_2} = 74 torr, pulmonary venous blood P_{O_2} = 312 torr, F_{IO_2} = 0.7). The addition of PEEP decreased Pa_{O_2} from 74 to 61 torr. This hypoxemia was independent of F_{IO_2}. During this period right atrial pressures were found to be higher than pulmonary venous pressure. The patient was weaned on IMV, with immediate improvement in Pa_{O_2} to 109 torr (F_{IO_2} = 0.7).

The left atrial line was withdrawn from the pulmonary vein

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(P_{O_2} = 230 torr) to the left atrium, where P_{O_2} was 119 torr. This suggested an intracardiac shunt, since there was no clinical or roentgenographic evidence of an abnormality in the right lung. Clinically, the patient was well. Following extubation of the trachea, he recovered, with no further complication. Anticoagulation with heparin was done to prevent thrombus formation with possible paradoxical emboli.

Discussion

Hypoxemia associated with reopening of a patent foramen ovale has previously been reported to follow CPB.[4,6,7] The incidence of this complication is unknown, but few patients following CPB have hypoxemia of the severity documented in these two cases. Our experience in the cardiac catheterization laboratory suggests that the incidence of a patent foramen ovale is considerably less than the 25 per cent reported by Edwards[8] at pathologic examination. During routine right-heart catheterization via the inferior vena cava, a Cournand catheter was passed into the left atrium in less than 10 per cent of our cases. Similarly, with the Brockenborough method, in spite of full "palpation" of the fossa ovalis, the catheter passed in less than 10 per cent of our patients. Documenting the presence of a patent foramen ovale preoperatively in patients about to undergo cardiac surgical procedures assists in the postoperative management of severe hypoxemia. Our present suggestion would be that digital examination of the septum at the time of right atrial cannulation would be warranted, and if a large open "flap valve" is found, corrective suturing should be considered.

In a previous study[9] of the relationship between right and left atrial pressures in patients following aortocoronary bypass procedures, 15 of 61 measurements showed P_{RA} higher than P_{LA}; thus, in this group of patients the physiologic situation for a right-to-left shunt frequently exists if the foramen ovale is anatomically patent.

The relationship between right and left atrial pressures is determined by the relative compliances, contractility, and loading of the two atria and ventricles. Each of these factors may be altered by cardiac surgery.

In another study[3] we demonstrated an increase in pulmonary vascular resistance (PVR) in many patients following CPB. This increase in PVR may be aggravated by the use of intermittent positive-pressure ventilation (IPPB) and PEEP. A rise in PVR can increase right venticular end-diastolic pressure (RVEDP) and volume (RVEDV) by increasing right ventricular afterload. Laver[10] has demonstrated that PEEP can cause enlarged RVEDV and elevated RVEDP that can be significant in patients with pulmonary hypertension secondary to acute respiratory failure. Thus, the effects of IPPB on the pulmonary vascular bed are increased by PEEP.[11] The presence of this pulmonary hypertension (and increased right ventricular afterload) in the postoperative period, when right ventricular dysfunction can occur, may lead to increased right atrial pressure. This increased right ventricular afterload can restrict the effectiveness of volume replacement, with further increases in P_{RA} as fluids are administered. Decreased compliance of ventricular muscle has been documented[12] following ischemic arrest and cardiopulmonary bypass. The duration of this decreased compliance varies considerably among patients and appears to be related to the duration of ischemic arrest, even when cold cardioplegic solutions are used, as in our cases. A decrease in right ventricular compliance could also contribute to elevated P_{RA} values at any given level of RVEDV.

The pressure relationship between right and left atria can also be altered by the operative correction of a valve defect that had placed a chronic volume load on the left ventricle preoperatively. Compensatory left ventricular hypertrophy and dilatation secondary to aortic regurgitation (as in Patient 2) can result in low left atrial pressure with an adequate cardiac output following valve surgery. Postoperative volume replacement may result in elevated P_{RA}, for the reasons outlined above, with normal or even low left atrial pressures, as this left ventricle does not fail when confronted with a volume load. In any patient with an anatomically patent interatrial communication, this physiologic situation can result in opening of the flap, with resulting hypoxemia.

The diagnosis of this cause of hypoxemia following CPB depends ultimately on dye-dilution curves and catheterization of the heart with measurements of oxygen saturation. This report illustrates how the fortuitous placement of the left atrial line into a pulmonary vein can be used to suggest the diagnosis if the line is used to sample pulmonary venous blood. Although sampling from one pulmonary vein is not equivalent to an integrated sample from both lungs, the extent of intrapulmonary shunt necessary to produce the profound hypoxemia documented in these two cases would surely have resulted in some radiologic[2] or clinical evidence of intrapulmonary disease. Moorthy *et al.*[13] recently documented the significance of blood-gas determinations in samples obtained from left atrial cannulas following CPB. A calculated shunt fraction of the magnitude demonstrated in the case of Patient 1 (approximately 50 per cent) following CPB is rare.[3] The proportion of the measured shunt (and associated hypoxemia) secondary to intrapulmonary ventilation–perfusion mismatching is impossible to determine precisely.

In similar patients without intracardiac shunting, measured shunt fractions in the immediate post-CPB period averages 12–17 per cent.[3]

Once the diagnosis is made, therapy can be directed toward decreasing right ventricular afterload and improving the contractility of the ventricle. Both patients showed increasing hypoxemia when PEEP was added. This may result from the decrease in cardiac output with a secondary reduction in mixed venous oxygen tension ($P\bar{v}_{O_2}$) following the addition of PEEP. The result of any decrease in $P\bar{v}_{O_2}$ is an increase in hypoxemia resulting from any degree of right-to-left shunt, whether this shunting is intrapulmonary or intracardiac. Cassidy *et al.*[11] have shown that the decrease in cardiac output during PEEP is associated with a rise in transmural right atrial pressure, while transmural left atrial pressure decreases or remains unchanged in normal subjects. Whether the increase in hypoxemia seen in our two patients after PEEP was the result of a decrease in $P\bar{v}_{O_2}$ (secondary to a reduction in cardiac output) with the same degree of shunt, or was secondary to an increase in the quantity of right-to-left shunting across the patent foramen ovale, must remain conjectural.

Other case reports[4,6,7] of reopening of a patent foramen ovale after CPB have stressed the frequency of paradoxical emboli. Similar physiologic situations have been reported to aggravate hypoxemia in chronic obstructive pulmonary disease,[14] acute myocardial infarction,[15] and pulmonary emboli[16] when right atrial pressure rises above left atrial pressure. Our two cases demonstrate that the outcome can often be uneventful; however, the danger of air[17] and particulate emboli[4] in the perioperative period should be emphasized.

In summary, the combination of decreased right ventricular contractility and compliance in the presence of an increase in right ventricular afterload can result in a rise in P_{RA} to above the pressure in the left atrium. The equalization[5] or reversal of the normal interatrial pressure gradient can open a patent foramen ovale, as illustrated by these two cases. The importance of documenting the presence of a patent foramen ovale prior to a cardiac surgical procedure is emphasized, and the value of bedside techniques in confirming the diagnosis illustrated. With appropriate management aimed at reversing the interatrial pressure gradient, both patients with hypoxemia recovered from their operations with no adverse sequelae.

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