Pulmonary dysfunction after coronary artery bypass surgery

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Respiratory dysfunction and arterial hypoxemia are common causes of morbidity following cardiac surgery utilizing extracorporeal circulation. Predisposing factors can be classified as (1) related to the surgical procedure such as opening the chest, opening the pleura, trauma, postoperative pleural effusion, hemothorax, or pneumothorax; (2) associated preoperative pathologic changes such as restrictive or obstructive lung disease, chronic lung congestion secondary to heart disease; and (3) related to cardiopulmonary bypass technique with its associated physiologic and histologic changes in the lung structure.

We studied the degree of respiratory dysfunction and the course of recovery in 357 consecutive patients who had undergone coronary artery bypass surgery and were considered clinically capable of supporting their own pulmonary functions without prolonged mechanical assistance after surgery. Postoperative pulmonary management was standardized; all patients were mechanically ventilated overnight. FIO_2 and ventilatory parameters were adjusted to maintain PaO_2 80 to 120 mm Hg and $PaCO_2$ 35 to 40 mm Hg. Patients who had vital capacity 10 to 15 cc/kg body weight, PaO_2 >80 mm Hg inspiratory negative Pr >30 cm H_2O were weaned from mechanical ventilation. All patients

received routine chest physiotherapy until the fifth postoperative day. Computer analysis was used to compare postoperative screening spirometry results done on the seventh postoperative day with preoperative values, which were used as controls. There was a significant reduction in vital capacity, forced vital capacity, forced expiratory flow rate, and peak expiratory flow rate (*Table 1*). The FEV_1/FVC ratio remained the same, indicating that the changes are mainly restrictive and not obstructive.

The same group of patients experienced hypoxemia while breathing room air postoperatively. In patients with normal blood gases preoperatively, a fall of 5 to 12 mm Hg in PaO_2 was noted in early postoperative periods and remained so for 4 to 5 days even with stable hemodynamic conditions and normal cardiac output.

Although hypoxemia was evident on the second postoperative day the CO_2 level and pH were normal and did not show any significant changes from preoperative values (*Table 2*). By the seventh postoperative day while PaO_2 levels returned to preoperative values, PCO_2 was significantly lower with pH showing respiratory alkalosis.

These changes can be explained on the basis of mismatching of ventilation and perfusion and increased shunting of venous blood into pulmonary vessels due to atelectasis as evidenced by the changes in lung volumes during the same period. Hyperventilation and low PCO_2 is a compensatory corrective mechanism for these changes.

The drop in vital capacity and PaO_2 is greater in the older age group, patients with previous lung disease, and those who have internal mammary artery anastomosis as part of the operation. Opening of the pleura during dissection of the internal mammary artery is another added factor causing more

Table 1. Preoperative and postoperative screening spirometry results in 357 patients with CABG

	Vital capacity (L)	Forced vital capacity (L)	Forced expiratory volume (L)	Forced expiratory flow (L/sec)	Peak expiratory flow (L/sec)	FEV_1/FVC
Preoperative	4.12 ± 0.89	4.05 ± 0.90	2.90 ± 0.72	2.75 ± 1.18	8.58 ± 2.07	0.710 ± 0.08
Postoperative 7th day	2.81 ± 0.80	2.76 ± 0.79	1.96 ± 0.59	1.93 ± 0.80	6.73 ± 2.02	0.709 ± 0.08
Percent decrease	33.4	31.8	36.1	32	22.8	...
Paired *t* test (*t* value) p value = $p < 0.001$	36.5	33.5	34.77	18.71	13.73	

CABG = coronary artery bypass grafting.

Table 2. Changes in blood gases in first 7 days; 357 patients

	Preop	Postop day 2	p value	Postop day 7	p value
PaO_2 (room air)	76.03 ± 11.0	58.32 ± 9.4	<0.001	71.05 ± 9.8	NS
PCO_2	39.59 ± 4.4	40.01 ± 4.22	NS	31.9 ± 4.1	<0.001
pH	7.41 ± 0.03	7.43 ± 0.02	NS	7.47 ± 0.02	<0.001

Table 3. Comparison of incidence of atelectasis and changes in vital capacity and PaO_2 in groups A and B

	Group A	Group B	p value
Drop in vital capacity (>combined median value)	44.9% (84)	56.9% (96)	$p < 0.05$
Drop in PaO_2 (>combined median value)	71.3% (134)	85.7% (145)	$p < 0.025$
Atelectasis	8.5% (16)	16% (27)	$p < 0.025$

Group A, 188 patients had saphenous vein grafting only.
Group B, 169 patients had saphenous vein grafting plus left internal mammary artery anastomoses.

atelectasis and a further drop in vital capacity and PaO_2 in the postoperative period.

When we compared a group of patients who had saphenous vein grafting with another group who had saphenous vein grafting and internal mammary artery anastomosis there was a significantly higher incidence of atelectasis and a greater drop in PaO_2 and vital capacity in the second group (*Table 3*).

The results of the study indicate that despite prolonged mechanical ventilation and continuous postoperative pulmonary care, patients who clinically had adequate pulmonary functions and did not require further mechanical support still had a significant drop in lung capacities mainly due to varying degrees of atelectasis that may remain as long as 7 days postoperatively. Patients with restrictive lung disease may not tolerate further reduction in lung volumes postoperatively and are susceptible to more complications such as difficulty in weaning from mechanical ventilation, atelectasis, accumulation of secretions, and subsequent infection.

Associated hypoxemia and shunting are evident in early postoperative periods accompanied by compensatory hyperventilation. Patients who have the pleura opened during the operation should be maintained on sufficient end-expiratory pressure during ventilatory management to increase functional residual capacity and maintain large lung volumes, since they are susceptible to atelectasis and shunting.