Atelectasis and Gas Exchange after Cardiac Surgery

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Background: Sometimes a high intrapulmonary shunt occurs after cardiac surgery, and impairment of lung function and oxygenation can persist for 1 week after operation. Animal studies have shown that postoperative shunt can be explained by atelectasis. In this study the authors tried to determine if atelectasis can explain shunt in patients who have had cardiac surgery.

Methods: Nine patients having coronary artery bypass graft surgery and nine patients having mitral valve surgery were examined using the multiple inert gas elimination technique before and after operation. On the first postoperative day, computed tomography scans were made at three levels of the thorax.

Results: Before anesthesia, the average shunt was low (2 ± 3%; range, 0–13%), but on the first postoperative day shunt had increased to 12 ± 6% (range, 3–28%). The computed tomography scans showed bilateral dependent densities in all patients but one. The mean area of the densities was 8 ± 8% (range, 0–37%) of total lung area, corresponding to a calculated fraction of collapsed lung tissue of 20 ± 14% (range, 0–59%). In the basal region, the calculated amount of collapsed tissue was 28 ± 19% (range, 0–73%). One mitral valve patient was an outlier and had a large shunt both before and after the operation.

Conclusions: Large atelectasis in the dorsal part of the lungs was found on the first postoperative day after cardiac surgery. However, there was no clear correlation between atelectasis and measured shunt fraction. (Key words: Cardiopulmonary bypass; general anesthesia; lung physiopathology; mitral valve surgery; physiopathology; ventilation–perfusion ratio; x-ray computed tomography.)

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IMPAIRMENT of lung function, particularly oxygenation of the blood, is a common and potentially serious complication after cardiac surgery. It has been shown to persist for as long as 1 week after the operation.[1] Some proposed mechanisms are leukocyte activation,[2] atelectasis,[3] and fluid accumulation in the lung.[4] Few studies have examined atelectasis and shunt, and their correlation, after operation. After abdominal surgery, minor atelectasis has been found on the first postoperative day, whereas after lower limb surgery no atelectasis was found.[5,6]

Large atelectasis has been shown in the immediate postoperative period in patients with respiratory distress after cardiac surgery. Shunt correlated well with atelectasis.[7] In an animal study, Magnusson *et al.*[8] found large atelectasis with a corresponding shunt and hypoxemia, shortly after extracorporeal circulation. Animals subject to only sternotomy showed much less atelectasis. No difference in extravascular lung water was found between control, sternotomy, and animals subject to extracorporeal circulation.

Therefore we hypothesized that the shunt regularly seen in patients after cardiac surgery also can be explained by atelectasis formation.

In the current study, patients undergoing coronary artery bypass graft surgery (CABG) or mitral valve replacement or repair (MVR) were examined. These two categories were chosen in an attempt to get a wider range of pathophysiologic disturbances in the postoperative period, assuming that patients with mitral valve disease would show more atelectasis.

Our main purpose was to identify and to describe postoperative lung changes using computed tomography (CT) and to correlate findings with shunt, measured using the inert gas elimination method, and oxygenation.

Materials and Methods

We studied 18 patients who were scheduled to have either CABG (n = 9) or MVR (n = 9). Inclusion criteria

for the CABG patients were stable angina pectoris with left ventricular ejection fraction >40%. In all patients left internal mammary artery grafts were taken. The MVR patients had mitral regurgitation but no ischemic heart disease. One of them had a mixed lesion, and three had atrial fibrillation before operation. Patients with significant lung, kidney, liver, or cerebral disease were excluded from the study, as were patients with insulin-dependent diabetes mellitus, cardiac decompensation (New York Heart Association category IV), or other valve disease. In all patients routine static and dynamic spirometry and arterial blood gas analyses were done on the day before surgery.

The study was approved by the ethics committee of Uppsala University Hospital, and informed consent was obtained from each patient.

Anesthesia and Mechanical Ventilation

Morphine (7–15 mg) and scopolamine (0.3–0.6 mg) were given intramuscularly 1 h before anesthesia. Anesthesia and muscle relaxation was induced with 5–10 μg/kg fentanyl, 1.5–2.5 mg/kg thiopental, and 0.1 mg/kg pancuronium. After tracheal intubation, mechanical ventilation was started with 50% oxygen in nitrogen. Tidal volume (8–10 ml/kg) and respiratory frequency (10–14 breaths/min) were adjusted to maintain a normal arterial carbon dioxide tension. No positive end-expiratory pressure was used during the study.

Anesthesia was maintained with small doses of fentanyl (1 or 2 mg in total) and inhaled isoflurane (0.5–1 MAC). The lungs were not kept inflated during cold cardioplegic arrest. By the time the aortic cross clamp was removed, ventilation was resumed with 100% oxygen and one half the tidal volume of that before bypass. For the MVR patients, air was also expelled from the heart and central circulation by several deep breaths (approximate tidal volume, 1 l).

In the intensive care unit, mechanical ventilation was maintained as described before with an inspired oxygen fraction of 0.35–0.5, tidal volume of 8–10 ml/kg, and a respiratory frequency of 10–14 breaths/min. Patients were tracheally extubated when they were awake and hemodynamically stable, had a carbon dioxide tension <5.5 kPa (41 mmHg) while spontaneously breathing, an oxygen tension >10 kPa (75 mmHg), an inspired oxygen fraction <0.45, and a body temperature >37°C. All patients received 1–5 l/min supplementary oxygen until 20 min before the final measurements were made.

Cardiopulmonary Monitoring

Before induction of anesthesia a 20-gauge catheter was introduced in a radial artery and a triple-lumen, thermistor-tipped, 7.5-French pulmonary artery catheter was introduced *via* the right internal jugular vein into the pulmonary artery wedge position. Blood pressures relative to atmospheric pressure were measured. Electrocardiograph lead V5 was continuously recorded and used to calculate heart rate. Blood gases were determined by standard techniques (ABL 3; Radiometer, Copenhagen, Denmark), and oxygen saturation values were calculated according to standard formulas. Cardiac output was measured by thermodilution.[9,10] The alveolar–arterial oxygen tension difference ($PA-aO_2$) was calculated from the alveolar gas equation, assuming a respiratory quotient of 0.8.[9]

Measurement of Ventilation–Perfusion Distribution

The multiple inert gas elimination technique was used to assess the ventilation–perfusion distribution. The technique has been described before in detail.[11-15]

Intrapulmonary shunt (Q_S/Q_T) was defined as the fraction of total blood flow perfusing units with V_A/Q <0.005, "low V_A/Q" was defined as the fraction of blood flow perfusing units with V_A/Q between 0.005 and 0.1, "high V_A/Q" was defined as the fraction of ventilation to units with V_A/Q between 10 and 100, and dead space (V_D/V_T) was defined as the fraction of ventilation to units with V_A/Q >100. The dispersion around the mean V_A/Q ratio of the perfusion was defined as the logarithmic standard deviation of the perfusion distribution ($LogSD_Q$).

Computed Tomography

In all patients, CT (Somatom Plus, Siemens) was made 20–24 h after surgery. A frontal scout view of the thorax was obtained for positioning, and thereafter three axial scans were taken approximately 4 cm from each other. The first scan was positioned 1 cm above the top of the diaphragm, the second 2 cm below, and the third 2 cm above the carina. The scans were made after inspiration with a normal tidal volume of approximately 0.4 to 0.5 l. The scan time was 1 s at 255 mAs and 137 kV. The slice thickness was 8 mm and the matrix 512 × 512.

To measure the dependent densities, reflecting atelectasis,[16,17] a magnified image (approximately 3×) was made selectively of the dorsal region of the right and left lung. The dorsal border between the atelectasis and

the thoracic wall was plotted manually with exclusion of any pleural fluid. The ventral border was identified with the region-of-interest program. The atelectatic lung within the region of interest was defined as volume elements (VOXEL) in the interval from −100 to +100 Hounsfield units (HU).[18] The lung parenchyma ventral to the atelectasis was measured by the region-of-interest program including VOXELs from −100 to −1,000 HU.

The average attenuation (in Hounsfield units) of the dependent densities and the lung parenchyma ventral to the densities were also measured. With the assumption that the atelectasis contains nearly 100% tissue and essentially no air, and the lung parenchyma ventral to the atelectasis contains a mixture of air and tissue, the fraction of the total lung tissue that is collapsed can be calculated (appendix 1). It was also assumed that expansion of the atelectasis would give the whole expanded lung the same average attenuation as the nonatelectatic lung had before expansion. The calculations were made individually for the right and left side on each CT scan. When the amount of lung tissue in the aerated region was calculated, tissue attenuation was set to zero (i.e., the same as water). This is probably a fair assumption because a large part of lung tissue is blood and blood vessels, with an attenuation just above zero.[6]

Atelectasis was measured by one of the investigators (G.W.) who was blinded to study group assignments.

Experimental Procedure

Measurements were made before induction of anesthesia and on the first postoperative day, 20–24 h after surgery. On both occasions the patients were in a supine position and spontaneously breathing air.

Statistical Analysis

All data were collected and analyzed using a statistical program (StatView, Abacus Concepts, Berkeley, CA) and are presented as mean ± SD. The significance of a difference was tested by analysis of variance for repeated measures. All factors were considered fixed. For shunt and low V_A/Q, the Wilcoxon signed rank and the Mann-Whitney U tests were used. Spearman's rank correlation coefficient (rho) was used to analyze relations between variables. A P value <0.05 was considered significant.

Results

Table 1 shows demographic data and preoperative spirometric values. Surgeries were uncomplicated, and

Table 1. Demographic Data and Preoperative Spirometry

	CABG	MVR	P Value
n	9	9	
Gender (M/F)	8/1	3/6	
Age (yr)	58.7 ± 8.9	65.0 ± 9.8	NS
Height (m)	1.77 ± 0.08	1.68 ± 0.10	0.04
Weight (kg)	85.2 ± 13.6	70.4 ± 11.0	0.02
BMI (kg/m²)	26.9 ± 3.0	25.0 ± 3.0	NS
FVC% norm	90.4 ± 14.8	83.1 ± 16.2	NS
FEV1% norm	90.6 ± 11.4	87.9 ± 15.5	NS

Data are mean ± SD.

BMI = body mass index; FVC% norm = forced vital capacity (% of normal); FEV1% norm = forced expiratory volume in 1 s (% of normal); NS = not significant.

all patients were successfully extubated 5–12 h after extracorporeal circulation (ECC). Six patients in the MVR group and five in the CABG group needed small doses of ephedrine (70–300 μg/kg) or phenylephrine (1–2 μg/kg) for separation from ECC. No patient received a continuous infusion of inotropic drugs.

Hemodynamics and Gas Exchange

Table 2 shows hemodynamic and gas exchange results. Before induction of anesthesia, the arterial oxygen tension and alveolar-arterial oxygen tension gradient (PA−aO₂) were both normal.[19] On the first postoperative day, arterial and venous oxygen tensions were both significantly decreased, and PA−aO₂ increased, compared with control values.

Ventilation–Perfusion Relations

The fit of the ventilation and perfusion distributions to the raw data of excretions and retentions was good. All data are presented in table 3.

Before induction of anesthesia, shunt showed low values, but on the first postoperative day shunt had increased to a mean of 12 ± 6% in the whole material. One MVR patient (number 1) was an outlier and had a shunt of 13% before surgery and 28% on the day after surgery.

Blood flow through units with low V_A/Q was low, both before and after the operation.

Lung Densities

Mean attenuation in the dependent densities was 5 ± 16 HU (range, −43 to 36 HU). In the lung parenchyma ventral to the dependent densities, mean attenuation was −694 ± 83 HU.

Table 2. Hemodynamics and Oxygenation

	Awake		20 h after Surgery	
	MVR	CABG	MVR	CABG
HR (beats/min)	72 ± 11	56 ± 7	83 ± 18	83 ± 12*
MAP (mmHg)	100 ± 17	103 ± 7	78 ± 6*	77 ± 11*
MPAP (mmHg)	30 ± 14	23 ± 5	21 ± 5	17 ± 3†
PCWP (mmHg)	24 ± 16	16 ± 3	11 ± 4	9 ± 3*
CI (L·min^{-1}·m^{-2})	2.2 ± 0.6	2.4 ± 0.5	2.6 ± 0.7†	3.1 ± 0.4*
Pa$_{O_2}$ (kPa)	9.8 ± 2.2	10.0 ± 0.9	7.7 ± 1.3*	8.0 ± 0.9*
(mmHg)	(74 ± 16)	(75 ± 7)	(58 ± 10)	(60 ± 7)
Pv$_{O_2}$ (kPa)	4.6 ± 0.7	4.9 ± 0.4	3.7 ± 0.4*	4.2 ± 0.4*
(mmHg)	(35 ± 5)	(37 ± 3)	(28 ± 3)	(31 ± 3)
PA-a$_{O_2}$ (kPa)	3.0 ± 2.4	2.5 ± 1.1	5.4 ± 1.1*	5.1 ± 1.2*
(mmHg)	(22 ± 18)	(19 ± 8)	(41 ± 8)	(38 ± 9)

Data are mean ± SD.

HR = heart rate; Pa$_{O_2}$ = arterial oxygen tension; CI = cardiac index; Pv$_{O_2}$ = mixed venous oxygen tension; PA-a$_{O_2}$ = alveolar arterial oxygen tension gradient; MAP = mean systemic arterial pressure; PCWP = pulmonary capillary wedge pressure; MPAP = mean pulmonary arterial pressure.

* $P < 0.01$ *versus* control.

† $P < 0.05$ *versus* control.

Twenty hours after surgery all patients except one (number 5 of the MVR group) showed dependent densities on CT examination, with an average of 8 ± 8% of total lung area. The average for the MVR patients was 10 ± 11% and for the CABG patients it was 6 ± 4% (not significant). The average above and below the carina, including both groups, was 4 ± 7% and 7 ± 7%, respectively (fig. 1). The densities were largest in the basal scans (12 ± 12%) and diminished gradually toward the apex of the lungs ($P < 0.0001$).

When analyzing the right and left hemithorax, we observed significantly ($P = 0.01$) more densities in the left lung of the CABG patients. No such difference was observed in patients in the MVR group (table 4).

The calculated fractions of collapsed lung tissue to total lung tissue for the three scans together were 19 ± 10% for the CABG patients and 21 ± 18% for the MVR patients. In the scans above and below the carina, 8 ± 13% and 19 ± 13%, respectively, were collapsed. In the basal scans, as much as 28 ± 19% of the lung tissue was collapsed. No significant differences between the groups were found.

Table 3. Multiple Inert Gas Elimination Data

	Awake		20 h after Surgery	
	MVR	CABG	MVR	CABG
Q$_S$/Q$_T$ (%)	3 ± 4	2 ± 1	10 ± 8*	13 ± 5*
	(0–13)	(0–3)	(3–28)	(7–20)
Low V$_A$/Q (%)	2 ± 2	2 ± 2	0 ± 1	0 ± 1
	(0–5)	(0–5)	(0–2)	(0–2)
V$_D$/V$_T$ (%)	41 ± 8	38 ± 8	37 ± 4†	30 ± 6‡
	(33–51)	(24–49)	(33–44)	(24–39)
LogSD$_Q$	0.73 ± 0.31	0.72 ± 0.25	0.56 ± 0.20	0.53 ± 0.15
	(0.39–1.21)	(0.34–1.01)	(0.40–1.01)	(0.31–0.83)

Data are mean ± SD (range within parentheses).

Q$_S$/Q$_T$ = inert gas shunt; Low V$_A$/Q = fraction of blood flow to units units with V$_A$/Q between 0.005 and 0.1; V$_D$/V$_T$ = inert gas dead space; Log SD$_Q$ = logarithmic standard deviation of perfusion.

* $P < 0.01$ *versus* control.

† $P < 0.05$ (CABG *vs.* MVR).
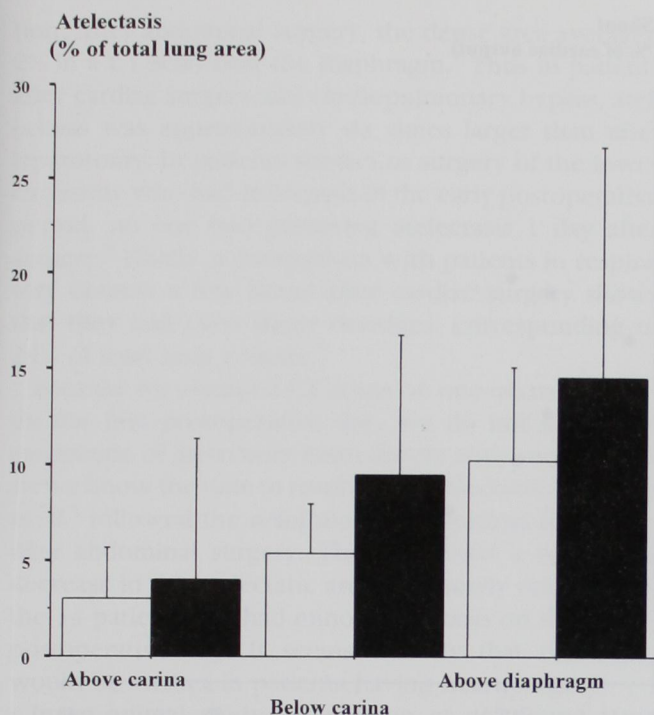
‡ $P < 0.05$ *versus* control.

Fig. 1. Dependent densities in the mitral valve surgery patients (*marked bars*) and the coronary bypass graft patients (*open bars*). Error lines indicate the confidence interval.

There was no clear correlation between dependent densities and shunt (fig. 2) for the whole material on the first postoperative day (rho = 0.45, P = 0.06). An individual analysis of the two groups showed a similar absence of correlation for the CABG group (rho = 0.05, not significant), whereas a high correlation was seen for the MVR patients (rho = 0.95, P < 0.01). One MVR patient (number 1) was an outlier, but the correlation was still significant if that patient was excluded from the analysis (rho = 0.93, P = 0.014).

No correlation between densities and oxygenation measured by $PA-aO_2$ and arterial oxygen tension was found for either group or when the groups were pooled.

Discussion

The main finding of our study is that, on an average, large densities in dependent lung regions were seen in 17 of 18 patients on the first day after cardiac surgery.

The pulmonary densities are interpreted as atelectasis.[17,20] There was less atelectasis in the cuts through the lung near the apex and the carina and more in the cut near the diaphragm. In the latter level, the dense area in four patients exceeded 20%, in one of whom 50% of the lung area was atelectatic.

Taking into consideration that the dense areas comprise approximately three to four times more lung tissue per VOXEL than the aerated region, as much as 30–70% of total lung tissue in the basal scan was atelectatic in 10 of the 18 patients on the first day after the operation. The assumption of three or four times more tissue in the dense areas is based on the average attenuation in the two regions. The aerated region had an attenuation (−694 HU) well in accordance with the mean lung attenuation previously found in healthy persons at functional residual capacity,[21] whereas the dense areas had an average attenuation very close to 0 HU, indicating complete airlessness.

Apart from a few deep breaths in patients in the MVR group when the aortic cross clamp was removed, no recruitment maneuvers or "sighs" were used to manage ventilation.

Errors in the Analysis of Atelectasis

A few factors that may have confounded our results should be mentioned. When patients were examined

Table 4. CT Results

	MVR		CABG	
	Right Side	Left Side	Right Side	Left Side
Dependent densities* (%)	7.4 ± 6.7 (0–23.1)	13.9 ± 18.3 (0–57.2)	3.7 ± 2.8 (1.2–8.4)	10.4 ± 7.6† (0–23.8)
Dependent densities‡ (cm²)	20.6 ± 16.6 (0–57.8)	24.2 ± 29.9 (0–94.2)	14.0 ± 9.7 (4.4–31.5)	27.4 ± 19.3 (0–51.7)

Dependent densities measured by computed tomography, average for the three scans together (above and below the carina and above the diaphragm). Data are mean ± SD (range within parentheses).

* Ratio of dependent densities to total lung area.

† P = 0.01 (right side *vs.* left side).

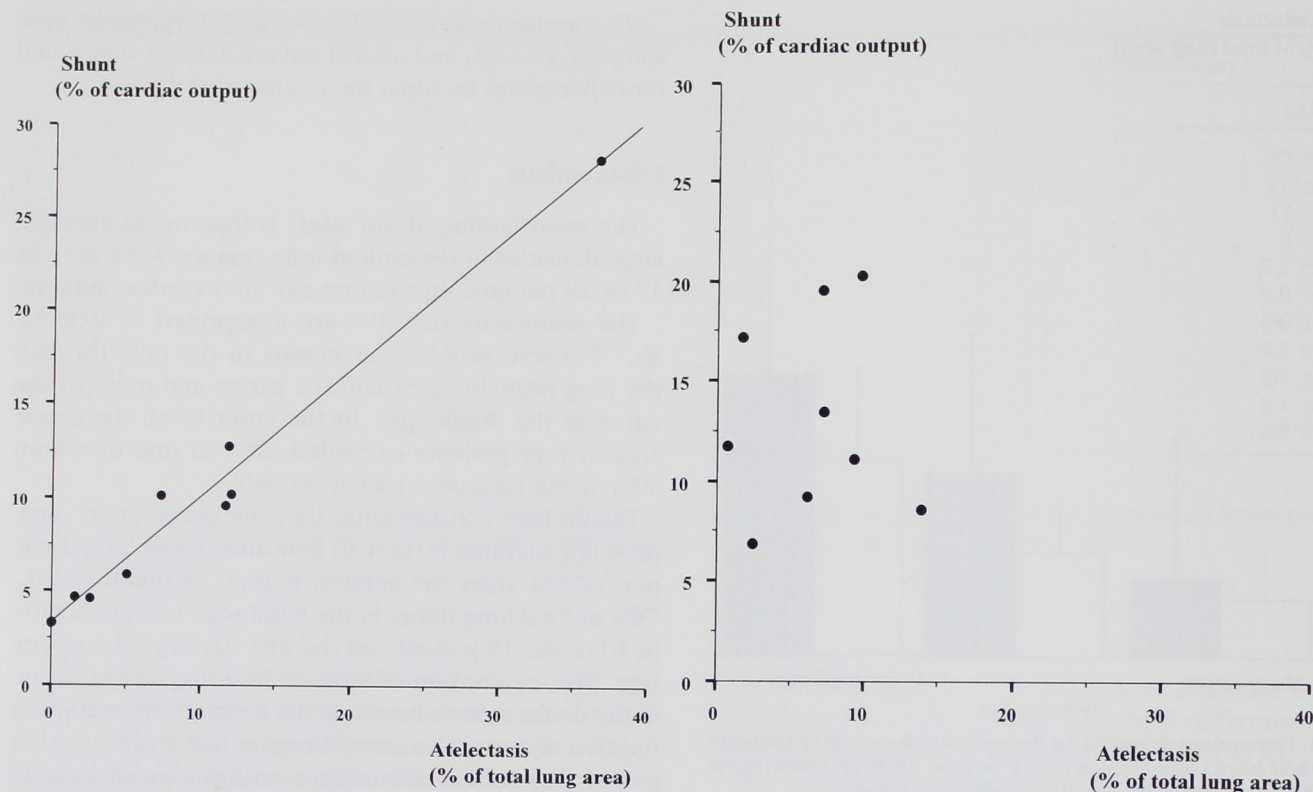‡ Dependent densities in absolute value.

Fig. 2. Correlation between atelectasis and pulmonary shunt on the first postoperative day. There was a significant correlation for the mitral valve patients (*left*); Spearman rho = 0.95, $P < 0.01$. No significant correlation was seen for the coronary artery bypass graft patients (*right*).

by spiral CT after induction of anesthesia, a continuous layer of atelectasis in the dorsal part of both lungs was found.[22] The atelectasis showed a progressive decrease from the diaphragm to the apex. In our study, the size of the atelectasis was calculated based on only three cuts, supposed to represent the whole lung. The results from the spiral CT study lend some support for this concept, but the calculation is still subject to considerable uncertainty.

In the current study we examined the patients after inspiration of an ordinary tidal volume when the CT scans were taken. This is in contrast to many previous studies of nonoperated patients in whom the scans were taken at functional residual capacity.[23,24] The change in protocol was made because many patients had difficulty holding their breath at expiration, but less so after inspiration. Rothen *et al.*[25] showed, in a study on anesthetized patients, a reduction of atelectasis with an inhalation of approximately 2.2 l, whereas the inhalation of a tidal volume, or even a double tidal volume, had no effect. Probably atelectasis was unaffected by the inspiration in our study. If it was affected, atelectasis

would have been even larger at functional residual capacity.

After lung collapse, the volume of the atelectatic lung tissue may change. Blood flow, and probably blood volume, are reduced because of hypoxic pulmonary vasoconstriction.[26] Flow in lymphatic vessels may be impeded and can affect extravascular lung water. These possible effects have not been accounted for in the calculations and may, to some extent, affect the results.

Finally, the mixture of crystalloid used for irrigation during the operation and shed blood may not have been completely evacuated through the pleural drains when the CT scans were taken. This mixture has an attenuation similar to that of atelectasis, and it can be difficult to separate the areas. In most cases, a border was seen and much effort was spent to distinguish between atelectasis and pleural fluid.

Comparison with Previous Studies

The atelectasis in the current study was considerably larger than previously seen in patients after abdominal and lower extremity surgery on the first day after opera-

tion. After abdominal surgery, the dense area averaged 2% in a CT scan near the diaphragm.[5] Thus in patients after cardiac surgery and cardiopulmonary bypass, atelectasis was approximately six times larger than after laparotomy. In patients subject to surgery of the lower extremity who had atelectasis in the early postoperative period, no one had persisting atelectasis 1 day after surgery.[6] Finally, a comparison with patients in respiratory distress a few hours after cardiac surgery shows that they had even larger densities, corresponding to 24% of total lung volume.[7]

Because we obtained CT scans on one occasion only, on the first postoperative day, we do not know the magnitude of atelectasis immediately after surgery, nor do we know the time to resolve the atelectasis. Lindberg *et al.*[5] followed the resolution of atelectasis for 4 days after abdominal surgery. They observed a successive decrease in the atelectatic area, but nearly one third of the 14 patients still had minor atelectasis on the fourth postoperative day. It seems unlikely that resolution would be swifter in patients having heart surgery.

In an animal study, Magnusson *et al.*[8] found large atelectasis shortly after ECC, whereas a group of animals who only had sternotomy had minor atelectasis. In the current study, and in previous studies by our group, the lungs were left nonventilated at zero positive end-expiratory pressure during ECC and the highest levels of shunt were also found shortly after ECC.[11,27]

No previous studies of patients having cardiac surgery have used CT scanning to assess atelectasis after operation. Regular chest radiographs underestimate both the frequency and amount of atelectasis. Our results and those from studies using regular chest radiographs cannot be directly compared.

Coronary Artery Bypass versus Mitral Valve Surgery

The amount of atelectasis was similar for the CABG and MVR patients, although the groups were too small to allow a definite conclusion.

The absence of a correlation between shunt and atelectasis for the whole material is similar to what was seen in patients who had undergone abdominal surgery,[5] but it is different from the good correlation that has been found during anesthesia.[23,24]

The difference between the anesthesia and postoperative periods is not clear, but return or strengthening of hypoxic pulmonary vasoconstriction after operation may contribute. Inhalational anesthetics reduce hypoxic pulmonary vasoconstriction,[28] and so does in-

creased venous oxygen saturation and a high fraction of inhaled oxygen,[26] all of which are present during anesthesia. The samples on the first postoperative day, on the other hand, were taken without supplementary oxygen, and both arterial and venous saturations showed low values. Hypoxic pulmonary vasoconstriction therefore can be expected to be more efficient in diverting blood flow from the atelectatic areas after operation, so that no clear coupling between atelectasis and shunt can be seen.

It is more surprising that a good correlation was seen in the MVR patients 1 day after surgery. Whether this finding was by chance or was an effect of the different pulmonary vascular tone in this category of patients is not clear.[29] A larger group of MVR patients must be studied before a conclusion can be made. We must admit however, that the same magnitude of shunt and atelectasis after operation in both groups does not correlate with the reappearance of hypoxic pulmonary vasoconstriction in one group but not in the other.

Conclusions

In this study we showed the formation of large atelectasis in 17 of 18 patients on the first day after cardiac surgery. The amount of atelectasis and shunt was similar in patients who had undergone MVR or CABG surgery. In the MVR patients, atelectasis correlated well with shunt, but no such correlation was found in the CABG patients. Gas exchange correlated poorly with atelectasis. This may be attributed to additional causes of gas exchange impairment and different degrees of compensatory mechanisms that obscure the effect of atelectasis.

Appendix 1: Calculation of the Fraction Collapsed Tissue

The normal lung is in these calculations considered a mixture of tissue and air. Air has by definition the attenuation of −1,000 HU. Tissue, which includes vessels, lung structures, and blood have approximately the same attenuation as water (*i.e.*, 0 HU).[6]

The volume fraction (f) of tissue in the nonatelectic lung can be calculated as follows:

$$A_n \cdot 1 = A_{air} \cdot (1 - f) + A_t \cdot f \qquad (1)$$

A_n = Mean attenuation of lung ventral to the dependent densities
A_{air} = Attenuation of air
A_t = Attenuation of tissue.

If formula 1 is solved for f,

$$f = (A_n - A_{air})/(A_t - A_{air}) \qquad (2)$$

and if A_{air} is set to $-1,000$ and A_t is set to 0:

$$f = (A_n + 1,000)/1,000. \qquad (3)$$

When the volume of the atelectasis is known, the fraction (F) of atelectatic lung tissue to total lung tissue can be calculated as follows:

$$F = V_{at}/(V_{at} + f \cdot V_n) \qquad (4)$$

V_n = Volume of lung ventral to the dependent densities
V_{at} = Volume of atelectasis.

The calculated fraction F is presented as a percentage in Results.

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