A new de-airing technique that reduces systemic microemboli during open surgery: A prospective controlled study

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Objective: We have evaluated a new technique of cardiac de-airing that is aimed at a) minimizing air from entering into the pulmonary veins by opening both pleurae and allowing lungs to collapse and b) flushing out residual air from the lungs by staged cardiac filling and lung ventilation. These air emboli are usually trapped in the pulmonary veins and may lead to ventricular dysfunction, life-threatening arrhythmias, and transient or permanent neurologic deficits.

Methods: Twenty patients undergoing elective true left open surgery were prospectively and alternately enrolled in the study to the conventional de-airing technique (pleural cavities unopened, dead space ventilation during cardiopulmonary bypass [control group]) and the new de-airing technique (pleural cavities open, ventilator disconnected during cardiopulmonary bypass, staged perfusion, and ventilation of lungs during de-airing [study group]). Transesophageal echocardiography and transcranial Doppler continually monitored the air emboli during the de-airing period and for 10 minutes after termination of the cardiopulmonary bypass.

Results: The amount of air embolism as observed on echocardiography and the number of microembolic signals as recorded by transcranial Doppler were significantly less in the study group during the de-airing time ($P < .001$) and the first 10 minutes after termination of cardiopulmonary bypass ($P < .001$). Further, the de-airing time was significantly shorter in the study group (10 vs 17 minutes, $P < .001$).

Conclusion: The de-airing technique evaluated in this study is simple, reproducible, controlled, safe, and effective. Moreover, it is cost-effective because the de-airing time is short and no extra expenses are involved.

The majority of the currently used conventional de-airing techniques evolved early after the advent of open surgery.[1] However, it was after the incorporation of intraoperative 2-dimensional transesophageal echocardiography (TEE) that the quality of de-airing improved and the procedure became better controlled.[2-5] This improvement in quality came at the expense of an increase in the time required for de-airing.[6,7] Despite meticulous removal of all visible air in the left side of the heart, the air emboli after the termination of cardiopulmonary bypass (CPB) continues to be an unresolved problem, and these emboli may show on TEE for as long as 28 minutes or more.[7] The unpredictable and precipitous advent of these emboli makes its surgical management difficult. The main source for these air emboli as seen on TEE is the pulmonary veins. These residual emboli may lead to ventricular dysfunction, life-threatening arrhythmias, and transient or permanent neurologic deficits.[8-10]

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This study evaluates a new technique for surgical de-airing that aims at preventing or minimizing ambient air from entering the pulmonary veins and expels air emboli from the pulmonary veins using a gradual increase in cardiac output coupled with delayed and staged ventilation. The technique is based on the assumption that for de-airing to be complete it is necessary to divert the entire cardiac output through the pulmonary vascular bed during the de-airing process, which is seldom possible in the de-airing period. This technique does not involve the use of carbon dioxide or any other equipment.

MATERIALS AND METHODS

Patients scheduled for elective true left open surgery were selected prospectively and consecutively for this de-airing study. The following preoperative exclusion criteria were applied: history of carotid artery disease, chronic obstructive pulmonary disease, emphysema, previous thoracic surgery, and thoracic trauma. Patients who required left internal thoracic artery grafting were also excluded from the study. The following exclusion criteria were applied intraoperatively: accidental opening of pleural cavities during sternotomy, failure to wean from the CPB, and difficulties in obtaining adequate Doppler signals from the right middle cerebral artery. Patients satisfying inclusion and exclusion criteria were assigned alternately to a control group (10 patients) and a study group (10 patients). The hospital ethical committee approved the study, and a signed informed consent was obtained from all prospective patients.

The preoperative patient demographic data are summarized in Table 1. One surgeon (B.K.) performed all the operations, and 1 surgeon (F.A.) performed all intraoperative transcranial Doppler (TCD) monitoring. Intraoperative TEE monitoring and recording were performed by 2 senior

Abbreviations and Acronyms
CPB = cardiopulmonary bypass
LV = left ventricle
MES = microembolic signals
TCD = transcranial Doppler
TEE = transesophageal echocardiography

cardiologists (A.R. and C.M.) who were blinded to the de-airing technique used and the TCD monitoring.

The patients were anesthetized and monitored in a standard manner, including intraoperative TEE. The surgery was performed via a median sternotomy. The right atrium was cannulated for venous drainage, and arterial blood was returned to the ascending aorta, arch of the aorta, or brachiocephalic trunk. The CPB circuit used in all patients comprised a membrane oxygenator (Compact Flow EVO Phiso, Sorin Group USA Inc, Arvada, CO), an arterial filter (Cobe Centry, Sorin Group USA Inc), and polyvinylchloride tubing (silicone tubing in the pump heads). Roller pumps (Stöckert S3, Sorin Group USA Inc) and a heat exchanger (T3, Sorin Group USA Inc) were used. CPB was established with calculated blood flows of approximately 2.5 L/m^2 body surface area at normothermia, and patients were cooled to 28°C to 25°C as measured in the urinary bladder/tympanic membrane depending on whether replacement of the aortic arch was performed. In all patients, the left ventricle (LV) was vented through the apex using a 15F Polystan LV drainage catheter (Maquet, Solna, Sweden). Antegrade cold blood cardioplegia was used in all patients.

De-airing Technique

Control group. During CPB, the pleural cavities were left intact and patients were ventilated with a minute volume of 1 liter at a frequency of 5/minute with a positive end-expiratory pressure of 5 cm H_2O. After completion of the surgical procedure, the heart and lungs were passively filled with blood from the heart–lung machine, the left side of the heart was gently massaged, and the LV was vented continuously. Full ventilation was resumed, and the aortic crossclamp was released. The heart was defibrillated to sinus or pacemaker-induced rhythm. The LV preload was successively increased by reducing the venous return to the heart–lung machine, and de-airing was continued under TEE monitoring. The LV vent was reduced, and the heart was allowed to eject if after progressive decrease in air emboli no or occasional air emboli were observed in the left side of the heart on TEE together with good cardiac ejection and normal central hemodynamics (de-airing time before cardiac ejection, Table 2). The patient was weaned from CPB, and the LV vent was stopped and clamped in situ provided that TEE continued to show freedom from air emboli in the left side of the heart (de-airing time after cardiac ejection, Table 2).

Study group. The de-airing technique has been described by us in detail.[11] In short, before starting CPB both pleural cavities were exposed through small openings in both mediastinal pleurae. After CPB was begun, the patient was disconnected from the ventilator allowing both lungs to collapse. After completion of the surgical procedure, the aortic root was de-aired, and the aortic crossclamp was released. The heart was defibrillated to sinus or pacemaker-induced rhythm. Good cardiac contraction and normal central hemodynamics were established. LV preload was now successively increased by reducing the venous return to the heart–lung machine, and de-airing was continued through the LV vent under TEE monitoring. When no air emboli were observed in the left side of the heart, the patient was reconnected to the ventilator and the lungs were ventilated with half of the estimated minute volume using 100% oxygen and 5 cm H_2O positive end-expiratory pressure. The de-airing was continued, and when no more air emboli were observed in the left side of the heart, the lungs were ventilated fully and the heart was allowed to eject (de-airing time before cardiac ejection, Table 2). The patient was weaned from CPB, and the LV vent was stopped and clamped in situ provided that TEE continued to show freedom from air emboli in the left side of the heart (de-airing time after cardiac ejection, Table 2).

All patients were observed continually for air emboli and microembolic signals (MES) for 10 minutes after weaning from CPB using TEE and TCD, respectively. During this period the LV vent and CPB cannulae were left in place and clamped. After the 10-minute observation period, CPB was restarted to remove the LV vent followed by removal of the venous and arterial cannulae. The heparin was reversed with protamine, and the chest was closed in a routine manner. In the study group, the pleural drains were placed only if pleurae were widely opened for reasons other than de-airing.

TABLE 1. Patient demography and preoperative clinical data by group (values shown are median with upper and lower quartiles for continuous variables)

	Control group (n = 10)	Study group (n = 10)	*P* value
Age (y)	62 (50–72)	70 (50–74)	.61*
Male/female	7/3	6/4	1†
Weight (kg)	80 (68–86)	75 (60–87)	.51*
Height (cm)	177 (166–184)	170 (157–180)	.42*
Body surface area (m^2)	1.9 (1.8–2.2)	1.9 (1.7–2.1)	.40*
Preoperative plasma creatinine (μmol/L)	66 (64–89)	84 (76–107)	.21*
Preoperative plasma ASAT (μkat/L)	0.39 (0.34–0.46)	0.42 (0.37–0.48)	.66*
Preoperative plasma ALAT (μkat/L)	0.30 (0.20–0.45)	0.36 (0.24–0.42)	.94*

ASAT, Aspartate amino transaminase; *ALAT*, alanine amino transaminase. *Wilcoxon test. †Fisher's exact test.

Transesophageal Echocardiography

Directly after weaning from the CPB, the left atrium, LV, and ascending aorta were monitored continuously for 10 minutes by TEE (Philips HP Sonos 5500, Andover, MA) using a 3-chamber view for residual air. The echocardiogram for each individual patient was recorded on a video tape for subsequent detailed analysis. The severity of air emboli as observed on TEE was classified in 4 grades as follows: grade 0, no residual air emboli; grade I, air emboli observed in left atrium only during 1 cardiac cycle; grade II, air emboli observed simultaneously in the left atrium and LV during 1 cardiac cycle; and grade III, air emboli observed simultaneously in the left atrium, LV, and aortic root during 1 cardiac cycle. To assess the severity of air emboli in detail, the 10-minute observation period was further subdivided into 3 intervals of 3, 3, and 4 minutes. The LV was vented whenever the air emboli exceeded grade II.

Trans-cranial Doppler monitoring. After termination of the CPB, the right middle cerebral artery was monitored continuously for MES using a multifrequency TCD scanning (Doppler box; DWL, Singen, Germany) for the first 10 minutes. The probe was fixed transtemporally by a head brace, and all MES were counted on-line automatically. The detection level for MES was an increase in power of more than 10 dB above background level and an embolus blood ratio that lasted 4 ms or longer simultaneously in both 2.0 and 2.25 M frequency channels. The insonation and reference gate depths were between 50 and 60 mm, sample volume was 10 mm, filter setting was 150 Hz, power was 180 mW, and gain was 10. The multifrequency Doppler has a sensitivity of 98.6% and specificity of 97.2% for detection of MES and artifacts.[12]

Statistics

Frequencies of different variables were compared using Fisher's exact test. Continuous variables are presented as medians and quartiles, and the

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TABLE 2. Clinical intraoperative and perioperative data by group (values shown are median with upper and lower quartiles for continuous variables)

	n	Control group (n = 10)	Study group (n = 10)	*P* value
Surgical procedures:	20			
Aortic valve replacement		70% (7, 5 mechanical)	50% (5, 2 mechanical)	.65*
Aortic valve repair		30 % (3)	30% (3)	1*
Ross operation		0% (0)	10% (1)	1*
Ascending aorta replacement		50% (5)	50% (5)	1*
Aortic arch replacement		20% (2)	10 % (1)	1*
Aortic annulus enlargement		30 % (3)	10 % (1)	.58*
coronary artery bypass grafting		30 % (3)	40 % (4)	1*
CPB time (min)	20	142 (130–184)	142 (108–226)	.83†
Aortic crossclamp time (min)	20	101 (90–121)	114 (76–173)	.91†
Total de-airing time (min)	20	17 (13–20)	10 (8–11)	<.001†
De-airing time before cardiac ejection	20	6 (4–8)	4 (4–6)	.33†
De-airing time after cardiac ejection	20	8 (7–12)	4 (3–6)	<.001†
Ventilator time (h)	20	5.0 (4.0–6.1)	4.2 (3.6–4.9)	.39†
Intensive care unit stay (h)	19	22 (20–34)	23 (22–78)	.28†
Hospital stay (d)	19	7 (6–11)	9 (7–11)	.37†
Postoperative plasma creatinine (μmol/L)	19	82 (76–96)	88 (69–107)	.84†
Postoperative plasma ASAT (μkat/L)	19	0.86 (0.64–1.08)	0.87 (0.75–1.60)	.67†
Postoperative plasma ALAT (μkat/L)	19	0.41 (0.26–0.48)	0.38 (0.32–0.50)	.97†

CPB, Cardiopulmonary bypass; *ASAT*, aspartate amino transaminase; *ALAT*, alanine amino transaminase. *Fisher's exact test. †Wilcoxon test.

Wilcoxon's rank-sum test was used for comparison. For comparison of repeated measurements of TEE grades for 10 minutes after CPB termination, a nonparametric method was used and data were expressed as estimated relative marginal effects of TEE grades with 95% confidence intervals.[13]

RESULTS

All patients in this study satisfied the preoperative and intraoperative inclusion criteria. The control and study groups were statistically similar in terms of patient demography data (Table 1). All patients survived the surgical procedure except 1 patient in the study group who died of irreversible cardiac failure in the early postoperative period. In this patient, the weaning from CPB was uneventful despite long bypass and aortic clamping times (230 and 200 minutes, respectively). The cause of death was systemic thrombosis probably because of ongoing treatment with methotrexate for severe polymyalgia rheumatica and the intraoperative use of aprotinin. The remaining patients had no significant postoperative complications and were weaned from the ventilator in the first 12 hours. The intraoperative and perioperative clinical data are summarized in Table 2.

The postoperative ventilation times in both groups were short and similar. The duration of chest-tube drainage in the 2 groups was similar, and no patient in the study group required pleural drainage because of postoperative hemo- or hemopneumothorax. The postoperative chest x-rays were also similar in both groups.

In the study group, 9 patients (90%) had grade I or lower air emboli on TEE monitoring during the first 3-minute observation period and all patients were free from air emboli thereafter (Figure 1). In the control group, 9 patients (90%) had grade II or greater air emboli during the first 3-minute observation period ($P < .001$, Figure 2), 5 of whom (50%) continued to remain so in the last 7 minutes after the CPB observation period ($P < .001$, Figure 2). Furthermore, during the 10-minute observation period, the LV vent was not reopened in any patient from the study group compared with 8 patients (80%) in the control group in whom the LV was reopened at least once because of grade III air emboli ($P < .001$).

The total de-airing time was significantly shorter in the study group compared with the control group (10 vs 17 minutes, respectively, $P < .001$, Table 2), and this was mainly because of the reduction in the de-airing time after the LV was allowed to eject ($P < .001$, Table 2).

During the de-airing period, there was a significant reduction in the total number of MES in the study group compared with the controls ($P < .002$, Figure 3). This reduction in MES occurred mainly after cardiac ejection ($P < .001$, Figure 3). In the first 10 minutes after the CPB observation period, the number of residual MES in the study group were also significantly lower than in the control group ($P < .001$, Figure 3).

DISCUSSION

The importance of the pulmonary veins for cardiac de-airing was emphasized as early as 1965, and some of the maneuvers suggested then are still being used in clinical practice.[14] The Trendelenburg position, partial clamping of the ascending aorta, and venting of the ascending aorta are not always optimal in preventing systemic air embolization because velocity of the blood in the ascending aorta is high.[15,16] These systemic air emboli are especially

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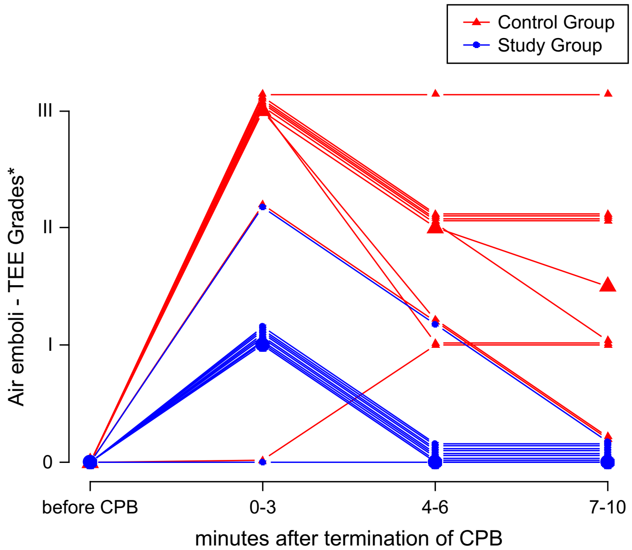

FIGURE 1. TEE 3-chamber view monitoring of air emboli during first 10 minutes after termination of CPB. *Grade 0, no residual air emboli; grade I, air emboli observed in 1 of the 3 anatomic areas during 1 cardiac cycle (left atrium, LV, aortic root); grade II, air emboli observed simultaneously in 2 of the 3 anatomic areas during 1 cardiac cycle; grade III, air emboli observed simultaneously in all 3 anatomic areas during 1 cardiac cycle. *TEE,* Transesophageal echocardiography; *CPB,* cardiopulmonary bypass.

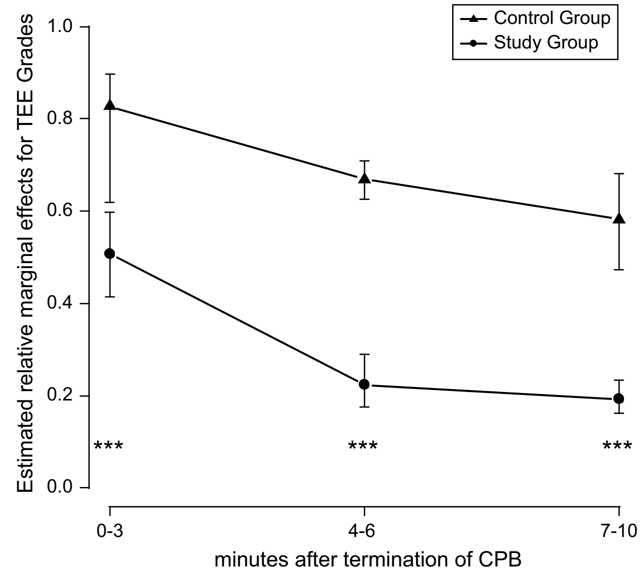

FIGURE 2. Statistical comparison of repeated measurements of TEE 3-chamber view grades of air emboli during first 10-minute observation period after termination of CPB. Data are expressed as estimated relative marginal effects of TEE grades with 95% confidence intervals.[13] ***$P < .001$. *TEE,* Transesophageal echocardiography; *CPB,* cardiopulmonary bypass.

undesirable in patients with early perioperative cardiac dysfunction. Moreover, the presence of intraoperative systemic air emboli has been shown to have a direct correlation to the postoperative neuropsychologic disorder.[17-20]

Our clinical interest in the prevention of systemic air emboli occurred several years ago after intrapericardial and bilateral clamping of the pulmonary veins. After total CPB was established, a vent was placed in the main pulmonary artery. After cardioplegia, the pulmonary veins were clamped. The pleural cavities were not opened, and patients were administered dead space ventilation during the period of cardioplegic arrest. After completion of the surgical procedure and release of the aortic crossclamp, the right side of the heart and the lungs were filled passively with blood and the pulmonary vein clamps were removed. The vent in the pulmonary artery was transferred to the LV. The heart was defibrillated and de-aired in a fashion similar to that discussed in this report. Although no proper clinical study was performed using this technique, the decrease in systemic air emboli as seen on TEE encouraged us to look for alternate simpler techniques that would prevent air from entering the pulmonary veins after the left side of the heart was exposed to ambient air.

Bilateral opening of the pleura equalizes the pressure in the pleural cavities to ambient atmosphere. When the lungs are disconnected from the ventilator, the induced pulmonary collapse decreases the amount of air entering the pulmonary veins after opening the left side of the heart. In addition, it is probable that the pulmonary veins, especially those from the anterior segments of both lungs, become kinked because of the posterior dislocation of the collapsed lungs. These mechanisms may prevent or minimize ambient air from being trapped in the pulmonary veins. In the early de-airing phase, when lungs are still in a collapsed state, a suboptimal right-

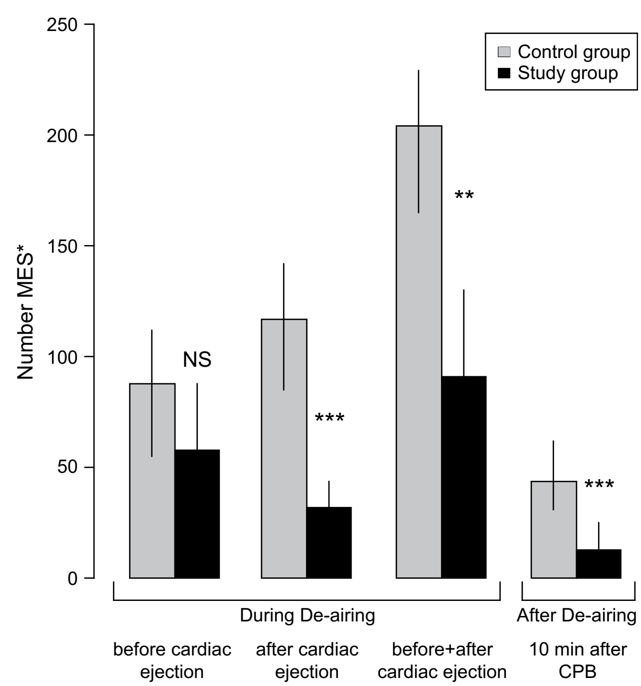

FIGURE 3. Number of MES as registered by TCD on right middle cerebral artery during de-airing and first 10-minute observation period after termination of CPB. *NS,* Not significant. ***$P < .001$. **$P < .002$. *MES,* Microembolic signals; *NS,* not significant; *CPB,* cardiopulmonary bypass.

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sided heart output is sufficient to fill the lungs completely with blood, thereby flushing out most of the trapped air from the pulmonary veins. Successive increase in the right ventricular pre-load and ejection coupled with delayed and concomitant increase in the minute volume ventilation contribute to keep the pulmonary veins free from the air until the patient is completely weaned from CPB.

The new de-airing technique reported here significantly reduced the de-airing time in the study group (10 vs 17 minutes, $P < .001$). Moreover, the freedom from residual air emboli (grade 0–I) as recorded by TEE was obtained in 90% of patients during the first 3 minutes after weaning from CPB compared with 10% of patients in the control group (Figures 1 and 2, $P < .001$ for all 3 time intervals). This technique also significantly reduced the number of MES as recorded by TCD in the first 10-minute observation period after the termination of CPB. These results are similar, if not better, to those described with carbon dioxide insufflation,[21] and this technique does not carry any risk of inducing acidosis, which may be the case with carbon dioxide insufflation.[22]

In this study, the LV was vented from the apex in all patients and left clamped in situ for the first 10 minutes of the observation period after CPB termination. This allowed intermittent venting of the heart in the event of grade II or greater air emboli showing on TEE. The LV vent was thus reopened in 8 patients (80%) in the control group during the first 3-minute post CPB observation period compared with none in the study group ($P < .001$). This would mean that the number of MES recorded on TCD in the control group is even higher than what is actually shown in Figure 3.

The number of residual MES recorded on TCD in the first 10-minute observation period after CPB correlated well with the TEE air emboli grades. The dual-frequency, multigated TCD was used in this study for automatic and on-line monitoring of the MES.

After the release of the aortic crossclamp and before cardiac ejection, the number of MES as recorded by TCD was similar in both groups. The number of MES decreased significantly in the study group first after the heart was allowed to eject (Figure 3). These findings indicate that the LV apical vent in combination with 1-time passive venting of the aortic root is not adequate for satisfactory de-airing of the aortic root itself. An active and more effective de-airing of the aortic root is therefore recommended before the aortic crossclamp is released.

The new de-airing technique evaluated in this study is based on achieving bilateral passive pulmonary collapse before the left side of the heart is exposed to the ambient air. Therefore, this technique may not be equally effective in patients in whom adequate pulmonary collapse cannot be achieved, that is, patients with chronic obstructive pulmonary disease, chronic pulmonary parenchymal diseases, previous pulmonary surgery, and other nonspecific pulmonary adhesions to the chest wall.

The CPB and aortic occlusion times in this study were long, whereas the postoperative ventilator times in both groups were relatively short with no significant difference between the groups. This suggests that bilateral pulmonary collapse per se does not negatively affect the early postoperative pulmonary function as judged by routine clinical respiratory parameters.

CONCLUSIONS

Our study shows that the integrated technique of bilateral-induced pulmonary collapse and successive increased filling of the lungs with a concomitant increase in mechanical ventilation during de-airing of the left side of the heart significantly reduces the number of systemic MES. This integrated technique also significantly reduces the de-airing time. Further, the systemic air emboli and MES are significantly reduced after the termination of CPB. The new de-airing technique that has been evaluated in a consecutive, prospective, and controlled manner is thus simple, short, controlled, safe, and effective. Moreover, it is cost-effective because the de-airing time is short and no extra expenses are involved.

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