

the authors aware of any evidence that this sealing of the pleural space has ever occurred, let alone that this action has been the mechanism behind the “clinical” success of all such procedures? The pleural changes reported at subsequent thoracotomy or postmortem examination, following previous “clinically successful pleurodesis,” have been clearly described and accepted; that is, pleural thickening associated with fibrin production, collagen deposition and fibrosis, plus variable adhesions. However, despite the very large number of “clinically successful pleurodeses” achieved over the last 80 years, there appear to be no similar reports demonstrating sealing or obliteration of the pleural cavity. It should be remembered that pleural adhesions do not prevent tethered pneumothoraces in the case of air leaks (or loculated effusions associated with pleural malignancy). In an age of evidence-based medicine, such an absence of evidence suggests a medical myth. Should the “success” of the procedure be more accurately attributed to those pleural changes that we know do occur?

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Reference

1. Dugan KC, Laxmanan B, Murgu S, Hogarth DK. Management of persistent air leaks. *Chest*. 2017;152(2):417-423.

The Surgical Point of View About Persistent Air Leaks Prevention First!



To the Editor:

We read with interest the paper by Dugan et al¹ recently published in *CHEST* (August 2017) about the management of persistent air leaks (PAL). We have appreciated the attention of *CHEST* regarding this complication that affects numerous pulmonary patients and which represents a daily challenge for thoracic surgeons. The review performed by the authors is exhaustive, including different aspects of

the problem and reporting the most popular therapeutic strategies to solve it.^{2,3} However, the first lesson learned in the operating theater is that “prevention is better than cure.” Thus, a careful intraoperative management of pulmonary parenchyma should be stressed because it represents the most efficient strategy to prevent PAL. The manipulation of the pulmonary parenchyma during surgery, in particular when marked pleural adhesions are present or in the case of emphysematous lungs, might be gentle to avoid air leakage.

One of the most important risk factors for the development of PAL in patients undergoing lobectomy is the absence of interlobar fissures, which eventuality obliges surgeons to work inside the parenchyma to reach the interlobar branches of the pulmonary artery, often creating minor damage to the remaining lobe. It could be resolved by using specific surgical techniques such as fissure-less lobectomy (the separation of fissures with mechanical staplers occurs after the resection of vascular and bronchial elements).⁴ This technique is almost always used during video-assisted lobectomy. In the case of lung volume reduction surgery or wedge resections performed in patients with other underlying pulmonary disease (eg, idiopathic pulmonary fibrosis), it could be helpful to use patches of pericardial tissue to wrap the loads of staplers and thus reinforce the staple lines. Furthermore, numerous sealants and glues are routinely used to cover lung parenchyma to avoid PAL, in particular when microlesions on the visceral pleural surface are present.⁵ Although these precautions do not eliminate the problem of PAL (they must be treated, as reported in the review¹), they dramatically decrease the incidence of PAL, with better postoperative recovery as well as a reduction in health costs.

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References

1. Dugan KC, Laxmanan B, Murgu S, Hogarth DK. Management of persistent air leaks. *Chest*. 2017;152(2):417-423.
2. Andreetti C, Venuta F, Anile M, et al. Pleurodesis with an autologous blood patch to prevent persistent air leaks after lobectomy. *J Thorac Cardiovasc Surg*. 2007;133(3):759-762.
3. Anile M, Venuta F, De Giacomo T, et al. Treatment of persistent air leakage with endobronchial one-way valves. *J Thorac Cardiovasc Surg*. 2006;132(3):711-712.
4. Anile M, Diso D, Rendina EA, Venuta F. A simple technique to avoid postoperative air leakages after right upper lobectomy. *Eur J Cardiothorac Surg*. 2013;43(4):e99-e100.
5. Venuta F, Diso D, De Giacomo T, Anile M, Rendina EA, Coloni GF. Use of a polymeric sealant to reduce air leaks after lobectomy. *J Thorac Cardiovasc Surg*. 2006;132(2):422-423.

Does the Time of Day a Pulmonary Embolism Response Team Is Activated Affect Time to Intervention or Outcome?



To the Editor:

In this letter, we evaluate how the activation time of the Pulmonary Embolism Response Team affects treatment decisions and time to intervention. Prior studies have demonstrated higher mortality rates for patients who originally presented with acute pulmonary embolism (PE) on a weekend as well as diverging survival curves after the first day of care, highlighting that early decision-making regarding treatment for PE may affect

30-day mortality.^{1,2} We evaluate how day and night groups, or “on-hours” and “off-hours” activations, could contribute to different treatment decisions as well as different times to intervention and 30-day mortality.

Of the 457 Pulmonary Embolism Response Team activations at the Massachusetts General Hospital, 317 occurred during the day and 140 during the night. **Table 1** describes the demographics of the patients as well as characteristics at time of presentation. The day and night patients were similar in, sex, PE severity, and other clinical characteristics.³ The data collection and analysis was approved by the Human Research Committee of Partners Health Care (protocol No. 2012P002257).

We found no difference in the rate of interventions within 24 hours of activation between the day and night groups (13% vs 15%; $P = .62$). Among those who received interventions within 24 hours, the distribution for the type of intervention was slightly different between the two groups: Catheter-directed therapy accounted for 81% of interventions in the night group but only 55% of interventions in the day group, and systemic IV thrombolysis and surgical embolectomy were more common in the day group ($P = .06$) (**Fig 1**). When limited to interventions that occurred within 6 hours (early intervention), the distribution of the type of intervention was similar between the two groups ($P = .99$). Among those who received interventions

TABLE 1] Characteristics of Day and Night Groups

| Variable | Day | Night | P Value |
|--------------------------------------------------|------------------|------------------|---------|
| All patients | n = 317 | n = 140 | ... |
| Age, mean (SD), y | 62 (16) | 61 (15) | .50 |
| Female sex, No. (%) | 140 (44) | 71 (51) | .20 |
| Race, No. (%) | | | |
| White | 266 (84) | 108 (77) | .16 |
| African American | 25 (8) | 13 (9) | |
| Asian | 4 (1) | 3 (2) | |
| Hispanic | 8 (3) | 2 (1) | |
| Other | 14 (4) | 14 (10) | |
| Highest heart rate, mean (SD), beats/min | 106 (22) | 107 (23) | .74 |
| Lowest systolic blood pressure, mean (SD), mm Hg | 115 (24) | 115 (21) | .76 |
| Highest respiratory rate, mean (SD), breaths/min | 22 (5) | 23 (7) | .14 |
| Troponin value, median (Q1-Q3), ng/mL | 0.03 (0.01-0.12) | 0.04 (0.01-0.11) | .30 |
| BNP value, median (Q1-Q3), pg/mL | 877 (215-3001) | 1313 (356-3810) | .93 |
| Simplified PESI score, median (Q1-Q3) | 1 (1-2) | 1 (0-2) | .81 |
| 30-day mortality, No. (%) | 36 (12) | 19 (16) | .32 |

BNP = brain natriuretic peptide, PESI = Pulmonary Embolism Severity Index.