

Hemodynamic and Pulmonary Changes after Drainage of Significant Pleural Effusions in Critically Ill, Mechanically Ventilated Surgical Patients

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Purpose: Our purpose was to study the effects of drainage of significant pleural effusions in mechanically ventilated patients in a surgical intensive care unit.

Methods: Twenty-two ventilated patients in the surgical intensive care unit of a tertiary care center over a 12-month period who developed a pleural effusion large enough to require drainage were studied prospectively. All patients underwent serial portable chest radiography in the upright or semiupright position; the radiographs were reviewed by a radiology attending. Pleural effusions were classified as small, moderate, or large. Moderate or larger effusions were drained using an 8- to 12-Fr pigtail catheter inserted at the bedside under ultrasound guidance. Hemodynamic and pulmonary parameters were collected before and after the fluid was drained. Parameters studied included those outlined in the physiologic

profile and included measured and calculated physiologic variables, arterial blood gas measurements, and SvO_2 measurements. Ventilator settings before and after were also recorded.

Results: Average initial pleural effusion drainage was $1,262 \pm 762$ mL (range, 300–2,980 mL). Nine of the 22 patients had effusions drained from both the right and left chest. Blood pressure, systemic vascular resistance, PO_2 , PCO_2 , SvO_2 , FIO_2 , peak airway pressure, and spontaneous volume did not change significantly. Pulmonary capillary wedge pressure decreased (17.4 ± 6.0 before, 13.6 ± 4.4 after; $p < 0.01$), central venous pressure decreased (14.2 ± 5.2 before, 11.5 ± 4.4 after; $p < 0.02$), and pulmonary arteriovenous shunt decreased (26.7 ± 15.1 before, 21.0 ± 7.8 after; $p < 0.04$). Oxygen delivery increased (579.7 ± 214.7 before,

662.8 ± 263.3 after; $p < 0.01$) and oxygen consumption increased (146.3 ± 61.6 before, 175.2 ± 73.8 after; $p < 0.01$). Respiratory rate also decreased (19.4 ± 6.5 before, 15.5 ± 6.3 after; $p < 0.05$). There were no complications from the placement of the pigtail catheters.

Conclusion: Drainage of pleural effusions results in increased oxygen delivery and oxygen consumption coinciding with a decrease in pulmonary capillary wedge pressure. The pulmonary arteriovenous shunt decreased, implying an increase in functional residual capacity and improved oxygenation. Further study is needed to determine whether these changes lead to an improved patient outcome (i.e., reduction in length of stay, ventilator days, or mortality).

Key Words: Pleural effusions, Hemodynamic and pulmonary changes.

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Although most patients are admitted to the surgical intensive care unit (SICU) for conditions other than pleural disease, the pleura is often involved by pulmonary parenchymal disorders and dysfunction of other organ systems. Most patients will have several chest radiographs during their SICU stay to detect pneumothorax and new infiltrates and to assess endotracheal tubes, chest tubes, and catheters. SICU patients appear to be at risk for developing pleural effusion because of the clinical setting and the severity of their primary disease. Many patients are admitted to the

SICU because of hypotension and hemodynamic instability and are treated with aggressive hydration, leading to fluid overload. Often, the patients are immobile because of pain, sedation, or paralytic drugs, thus placing them at risk for atelectasis. Occasionally, these effusions require drainage using large chest tubes or by performing thoracentesis. Although it is expected that draining the effusion would have a beneficial effect, a prospective study of the effects of drainage on the hemodynamic values and pulmonary physiology in patients admitted to the SICU has not been performed, until now. In addition, the insertion of relatively painless pigtail catheters performed with the use of ultrasound guidance was evaluated in this study.

PATIENTS AND METHODS

We prospectively studied patients admitted to the SICU of a tertiary care center for a 12-month period, who were on mechanical ventilation, had a pulmonary artery catheter in place, and developed pleural effusions large enough to require drainage. All patients were on volume-regulated ventilation (continuous mandatory ventilation or synchronized intermittent mandatory ventilation); no patients were on

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Table 1 Age, Sex, and Amount of Drainage of Plural Effusions

Patient	Left (mL)	Right (mL)	Total (mL)	Age/Sex
1	1,100	830	1,930	86/F
2	—	375	375	85/M
3	640	180	820	70/M
4	300	—	300	46/F
5	—	2,050	2,050	78/M
6	—	1,820	1,820	42/M
7	1,480	200	1,680	68/M
8	1,000	—	1,000	67/F
9	—	1,000	1,000	61/M
10	1,090	210	1,300	61/M
11	1,290	1,460	2,750	68/F
12	1,450	1,530	2,980	80/F
13	—	450	450	50/F
14	—	600	600	80/M
15	—	1,060	1,060	84/F
16	310	—	310	34/M
17	—	1,400	1,400	18/M
18	700	—	700	63/M
19	800	—	800	42/M
20	500	300	800	70/F
21	790	950	1,740	61/F
22	900	1,000	1,900	73/F
Average			1,262 ± 762	63 ± 18
Minimum			300	18
Maximum			2,980	86

Left, drainage from left side of chest; right, amount drained from right side of chest; total, total amount drained.

pressure-controlled ventilation. The data collected included age, sex, reason for admission to the SICU, complete blood counts, and serum electrolytes.

All patients underwent serial portable chest radiography in the upright or semiupright position. Each case was reviewed by a radiology attending. A pleural effusion was defined as small if the fluid obliterated the costophrenic angle or obscured the lower lung base, moderate if it opacified the lower and middle zones, and large if all three zones were opacified.

On the basis of clinical judgment, some of these patients underwent ultrasound or computed tomographic (CT) scanning as well. Those patients with pleural effusions estimated to be 300 to 500 mL or greater had their effusions drained. The drainage was carried out with an 8- to 12-Fr pigtail catheter inserted at the bedside or radiology suite under ultrasound guidance.

Measurements of multiple hemodynamic and respiratory parameters were obtained less than 1 hour before and 1 hour after the insertion of the pigtail catheter to drain each patient's pleural effusion. Primary hemodynamic parameters obtained via a pulmonary artery catheter were also recorded. These parameters included blood pressure, mean arterial pressure, pulmonary artery pressure, pulmonary capillary wedge pressure (PCWP), central venous pressure, cardiac output, pH, Pco₂, Po₂, HCO₃, Svo₂, and oxygen saturation. Respiratory parameters were obtained from each patient's ventilator including respiratory rate, peak airway pressure, positive end-expiratory pressure, pressure support, peak in-

spiratory pressure, minute volume, and spontaneous tidal volume. Using these data, physiologic profiles were generated for each patient both before and after catheter insertion. This facilitated derivation of cardiac index, stroke index, left ventricular stroke work, right ventricular stroke work, systemic vascular resistance, Cao₂, Cvo₂, arteriovenous oxygen difference, oxygen consumption, oxygen delivery, arteriovenous shunt, arterial base excess, and alveolar-arterial oxygen difference. The volume of fluid was recorded and a specimen was sent for analysis.

RESULTS

Twenty-two patients over a 12-month period were studied, ranging in age from 18 to 86 years (Table 1). All patients were mechanically ventilated and had pulmonary artery catheters in place. All were diagnosed with pleural effusions via chest radiographs. Eighteen (82%) had CT scan confirmation of the effusion, and four had ultrasounds (18%). A total of 30 catheters were placed in 22 patients (right, 36%; left, 23%; bilateral, 41%). The size of the catheters used were 8 Fr (85%), 10 Fr (10%), and 12 Fr (5%). Initial drainage ranged from 300 to 2,980 mL (mean, 1,262 ± 762 mL) of serous fluid (Table 1). All the drained fluid was negative for bacteria on Gram's staining and grew no organisms when cultured. There were no complications as a result of pigtail catheter placement in this series.

The 22 patients included in this study were all admitted to the surgical intensive care unit and had an average APACHE II score on the day of insertion of the catheters of

Table 2 Hemodynamic, Oxygenation, and Ventilator Parameters before and after Drainage of Pleural Effusions

Patients (n = 22)	Before Avg ± SD	Before Min-Max	After Avg ± SD	Paired t Test
SBP	126.3 ± 22.6	97–182	125.3 ± 21.5	NS
DBP	69.2 ± 19.2	41–106	66 ± 17.6	NS
MAP	88.2 ± 19.1	62.3–131.3	85.7 ± 17.1	NS
SPA	40.3 ± 11.5	23–60	38.7 ± 10.5	NS
DPA	22.2 ± 6.8	12–36	20.3 ± 5.2	NS
MPA	28.3 ± 8.0	16.3–42.0	26.4 ± 6.7	NS
HR	109 ± 19	73–150	107 ± 14.8	NS
RR	19.4 ± 6.5	11.0–36.0	15.5 ± 6.3	0.03
PCWP	17.4 ± 6.0	8.0–30.0	13.5 ± 4.4	0.003
CVP	14.2 ± 5.2	6.0–24.0	11.5 ± 4.4	NS
CO	7.7 ± 3.3	3.2–14.6	8.4 ± 4.0	NS
CI	4.3 ± 1.7	1.8–7.3	4.7 ± 2.0	NS
SVR	900 ± 495	356–1,765	841 ± 453	NS
SI	41.6 ± 14.7	17.0–74.7	43.9 ± 18.2	NS
LVS _W	46.2 ± 23.4	8.7–102.5	50.1 ± 19.3	NS
RVS _W	14.9 ± 6.2	6.6–29.1	15.2 ± 6.1	NS
TPR	1,588 ± 741	527–3,132	1,511 ± 782	NS
PVR	246 ± 159	75–704	271 ± 167	NS
SvO ₂	71.2 ± 10.7	45–88	71.5 ± 9.8	NS
SaO ₂	96 ± 4.5	81–99	97.2 ± 1.6	NS
CaO ₂	13.6 ± 1.8	9.4–17.3	14.4 ± 1.4	NS
CvO ₂	9.6 ± 2.1	5.9–14.0	10.5 ± 2.3	NS
AV-Diff	3.6 ± 1.4	1.5–7.1	3.9 ± 1.4	NS
QO ₂	146.3 ± 61.6	45–266	175.2 ± 73.8	0.004
Qs:Qt	26.6 ± 15.1	9.5–75.8	21.0 ± 7.8	0.03
Do ₂	579 ± 234.5	75.9–983.2	669 ± 268	0.01
A-a DO ₂	236 ± 170	32.7–580	211 ± 153	NS
Pco ₂	39.8 ± 7.3	29–57	38.3 ± 7.5	NS
PO ₂	96.5 ± 32.5	44–191	108.6 ± 23.5	NS
HCO ₃	25.2 ± 5.5	18–40	24.3 ± 5.1	NS
BE	0.6 ± 5.5	-5.1–15.3	-0.7 ± 4.8	NS
FiO ₂	0.45 ± 0.20	0.21–1.0	0.44 ± 0.16	NS
PaO ₂ /FiO ₂	245 ± 103	44–419	270 ± 101	NS
Tv	728 ± 69	600–870	722 ± 83	NS
PIP	34.9 ± 8.4	21–52	35.9 ± 12.5	NS
Sp MV	6.2 ± 2.5	1.2–10	6.6 ± 2.5	NS
Sp Tv	481 ± 138	190–630	606 ± 228	NS

Avg, average; Min, minimum; Max, maximum; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; SPA, systolic pulmonary artery pressure; DPA, diastolic pulmonary artery pressure; MPA, mean pulmonary artery pressure; HR, heart rate; RR, respiratory rate; PCWP, pulmonary capillary wedge pressure; CVP, central venous pressure; CO, cardiac output; CI, cardiac index; SVR, systemic vascular resistance; SI, stroke index; LVS_W, left ventricular stroke work index; RVS_W, right ventricular stroke work index; TPR, total peripheral resistance; PVR, pulmonary vascular resistance; SvO₂, mixed venous saturation; SaO₂, arterial oxygen saturation; CaO₂, arterial oxygen content; CvO₂, venous oxygen content; AV-Diff, arteriovenous oxygen difference; QO₂, oxygen consumption index; Qs:Qt, shunt; Do₂, oxygen delivery index; A-a DO₂, alveolar-arterial oxygen difference; Pco₂, arterial partial pressure of carbon dioxide; PO₂, arterial partial pressure of oxygen; HCO₃, arterial bicarbonate; BE, arterial base excess; FiO₂, fraction of inspired oxygen; PaO₂/FiO₂, PaO₂/FiO₂ ratio; Tv, tidal volume; PIP, peak inspiratory pressure; Sp MV, spontaneous minute ventilation; Sp Tv, spontaneous tidal volume.

17 ± 6 (range, 7–28) before the start of this prospective study. Six (27%) patients were admitted after blunt trauma, and 12 (55%) patients were admitted for treatment of post-operative abdominal sepsis. Four additional patients were being treated for necrotizing pancreatitis. All these patients showed compromised oxygenation, with an average PaO₂/FiO₂ ratio < 250.

PCWP significantly decreased with pigtail catheter placement from 17.4 ± 6.0 before to 13.6 ± 4.4 after drainage ($p < 0.01$) (Table 2 and Fig. 1). Central venous pressure also decreased significantly (before, 14.2 ± 5.2; after, 11.5 ± 4.4; $p < 0.02$) (Table 2 and Fig. 1). Similarly, shunt de-

creased and oxygen delivery index increased significantly (Table 2 and Fig. 3). Importantly, oxygen consumption also increased (Table 2 and Fig. 3). Likewise, respiratory rates decreased (before, 19.4 ± 6.5; after, 15.5 ± 6.3; $p < 0.03$) (Fig. 2). The other variables did not change significantly (Table 2).

DISCUSSION

There is normally a very small amount of fluid in the pleural space, probably less than 10 to 20 mL.¹ This fluid accumulates when pleural fluid formation exceeds pleural fluid absorption. Normally, fluid enters the pleural space

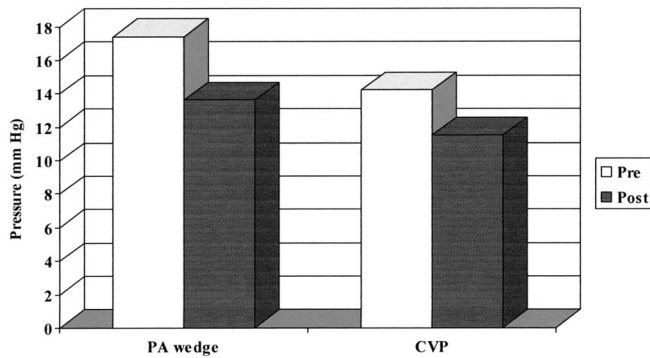


Fig. 1. Pulmonary artery capillary wedge pressure and central venous pressure before and after effusion drainage.

from the capillaries in the parietal pleura and is removed via the lymphatics situated in the parietal pleura. Fluid can also enter the pleural space from the interstitial spaces of the lungs via the visceral pleura or from the peritoneal cavity via small openings in the diaphragm. The lymphatics have the capacity to absorb 20 times more fluid than is formed. Accordingly, a pleural effusion may develop when there is excess fluid formation or when there is decreased fluid removal by the lymphatics.²

Patients in the intensive care unit accumulate pleural effusions from various causes such as heart failure, atelecta-

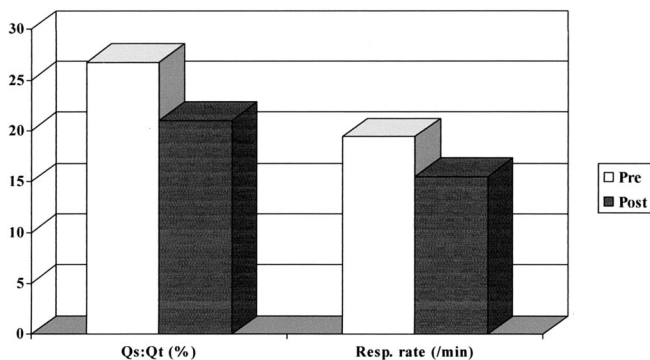


Fig. 2. Pulmonary arteriovenous shunt and respiratory rate before and after effusion drainage.

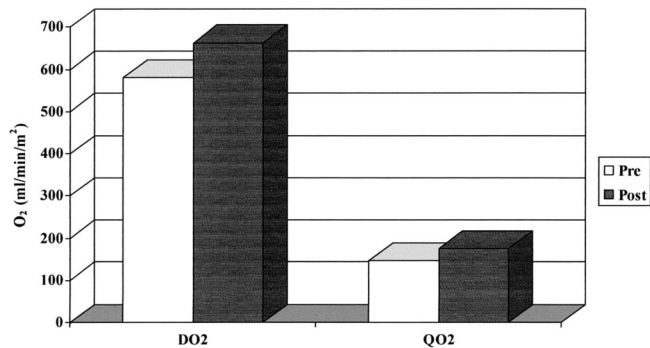


Fig. 3. Oxygen delivery and consumption before and after effusion drainage.

sis, pneumonia, pancreatitis, hypoalbuminemia, and so forth. They are rarely admitted to the intensive care unit for primary pleural disease. Patients may have a positive fluid balance, especially during the first few days of their intensive care unit stay, worsening alveolar-arterial oxygen gradient, and elevating pulmonary capillary wedge pressure. Atelectasis and alveolar collapse cause increased negative pleural pressure that theoretically would result in increased volume of interstitial ultrafiltrate moving into the pleural space. Atelectatic effusions result from severe muscle weakness, airway obstruction, restraints, or alteration of mental status as a consequence of the primary disease.³

A prospective study evaluating the hemodynamic changes before and after drainage of pleural effusions in an SICU has not been reported. Studies in dogs have confirmed that by infusing saline into the pleural spaces, intrapericardial pressure increases, resulting in tamponade-like physiology and echocardiographically demonstrated right ventricular collapse. This occurs because increased intrapleural pressure may be transmitted to the pericardial space, resulting in impaired cardiac filling and reduced stroke volume.⁴ In two case reports, Kaplan et al. showed that large pleural effusions can cause hemodynamically significant cardiac tamponade.⁵

Percutaneous catheter drainage of intrathoracic collections has developed as a natural extension of similar interventional radiologic procedures in the abdomen. The advent of CT scanning and sonography, which allow detection and characterization of pleural and parenchymal collections, combined with drainage catheter design and interventional techniques have made imaging-guided management of intrathoracic collections a safe and effective alternative to traditional surgical therapy.⁶

Placement of such a catheter using a Seldinger technique is virtually atraumatic.⁷ After an initial learning curve and with accumulated experience, radiologically placed catheters have been an efficacious treatment of pleural effusions and have a low complication rate.⁸ A special bedside technique using ultrasound guidance in critically ill patients can also be used.⁹

The increases in oxygen delivery can be tied to a combination of nonsignificant increases in both cardiac output and oxygen content in the arterial blood. Importantly, though, the oxygen consumption is also increased. This means the patients were on the critical portion of the oxygen delivery/oxygen consumption curve (oxygen delivery-dependent oxygen consumption). This implies at least in theory a beneficial improvement for the patient.

Improvement in the pulmonary arteriovenous shunt may be more difficult to interpret. Drainage of the effusions results in an increase in functional residual capacity. This restoration of functional residual capacity can open collapsed alveoli, leading to the decrease in the pulmonary arteriovenous shunt.

CONCLUSION

The patients who develop moderate to large pleural effusions in the SICU often have high filling pressures and pulmonary capillary wedge pressures. Drainage of these effusions results in increased oxygen delivery and oxygen consumption coinciding with a decrease in PCWP. The pulmonary arteriovenous shunt decreased, implying an increase in functional residual capacity and improved oxygenation. This study is limited in only considering short-term physiologic changes and not long-term outcome. Further studies are needed to determine whether these changes lead to improved patient outcome (i.e., reduction in length of stay in the intensive care unit, ventilator days, or mortality).

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