WBCs, 80 percent of which were mononuclear cells. Pleural fluid Gram stain showed Gram-positive cocci in short chains with a few Gram-negative rods. Culture of the pleural fluid grew 30 percent *Bacteroides melanogenicus*, 40 percent *Streptococcus viridans*, and 30 percent *S. group C*. Blood cultures were negative.

A chest tube was inserted into the left anterior axillary line, fifth intercostal space, on the evening of admission by the surgical resident. The tube placement was noted to be traumatic, since several attempts were required for placement. At the time of insertion, pus was observed escaping from the thoracostomy site. Initially, over the first eight hours, approximately 1,500 ml of purulent material was drained into an Emerson system. A small amount of subcutaneous air was noted at the chest tube insertion site. The patient remained stable for three days while being treated with 4 million units of aqueous penicillin *G* every six hours, although she continued to develop intermittent fevers. On the fourth hospital day an area of erythema and tenderness was seen extending from the chest tube entrance site into the axilla to the area overlying the left iliac crest. Cutaneous needle aspiration for culture was done, but grew no organism. Gentamycin was empirically added to the patient’s antibiotic coverage, but the patient continued to deteriorate, with further spread of the cutaneous inflammatory response. At the same time a pneumothorax thought to be due to a bronchopulmonary fistula developed, necessitating a second chest tube on the left anteriorly.

On the eighth hospital day, the patient suffered respiratory arrest with subsequent seizure. Chest roentgenogram showed a diffuse five-lobe alveolar infiltrate thought to represent the adult respiratory distress syndrome. On the morning of the ninth day the intubegulatory involvement had encompassed an area from the umbilicus around the left side to the midline of the back with obvious crepitation. A clinical diagnosis of necrotizing fasciitis was made. Radical incision and drainage of both flanks with placement of drains was performed. Purulent, foul-smelling material was present with extensive undermining of fascial planes. The patient continued to deteriorate, with hypotension and evidence of acute renal failure. On the tenth hospital day, the patient suffered further hypotension and died.

**DISCUSSION**

We have described a case of necrotizing fasciitis which, to our knowledge, is the first reported case occurring after tube thoracostomy for drainage of an empyema. Necrotizing fasciitis is a rapidly progressing infection of subcutaneous tissue or fascia which is often accompanied by severe systemic toxicity. Classic hallmarks of the disease, as seen in our patient, include the rapid development of spreading erythema, edema, warmth, and tenderness, with secondary systemic toxicity. The course of infection is variable, but reported to occur after trauma or surgery. The skin trauma may be as insignificant as an insect bite, and frequently no history of trauma can be elicited. We believe that our patient’s necrotizing fasciitis was directly related to the chest tube placement, since the infection originated in the area of the chest tube with no other source of infection being present. Additionally, although NF was initially described as being due to *S. pyogenes*, Giuliano and associates showed the most likely causative organisms to be anaerobic bacteria, facultative anaerobic, enterobacterial and non-group A streptococci. Our patient had anaerobic organisms in the pleural fluid which we believe were most likely the cause of her NF. Why NF developed as a complication of tube thoracostomy is unknown, but we speculate that the difficult tube insertion may have played a role in the subcutaneous dissemination of the organisms.

The critical period for diagnosing NF is within the first 24 to 48 hours after onset. In addition to antibiotics, treatment includes surgical debridement to remove devitalized tissue and to restore an aerobic environment. The procedure is dramatic, with wide incisions and drainage; possibly amputation may even be needed to curb the spread of necrosis. From the surgical standpoint, there is reluctance to use such invasive and mutilating techniques on a simple “cellulitis,” but prior experience has indicated that this is necessary to achieve any success. Recovery and rehabilitation may be long and complex, especially if the process involves any part other than an extremity. A critical aspect of diagnosing NF remains a high index of suspicion, especially when simple cellulitis becomes more severe. The diagnosis of NF must be considered when clinical findings of spreading cellulitis occur after tube thoracostomy.

**REFERENCES**


**Contralateral Effusions Secondary to Subclavian Venous Catheters* Report of Two Cases**

*Laurence M. Ciment, M.D.; Abraham Rotbart, M.D.; and Robert N. Galbut, M.D.*

Two cases of contralateral pleural effusions due to indwelling central venous catheters are presented. Radiographic contrast studies were performed to elucidate diagnosis and to define the mechanism of this complication; mediastinal leakage was documented in one case.

Since the introduction of central venous catheterization for fluid infusion, pressure monitoring, and hyperalimentation, the literature had been replete with reports of serious and mortal complications. These include pneumothorax, pleural effusion, pulmonary embolus, retroperitoneal bleed, and contralateral pleural effusion. It is the purpose of this report to document two cases of contralateral pleural effusion secondary to subclavian venous catheterization.

**CASE REPORTS**

**Case 1**

A 64-year-old female with a history of congestive heart failure and hypertension was admitted for medical management. The patient had been admitted to another hospital for acute dyspnea and was noted to have hypoxemia and hypotension. A right subclavian vein tunneled catheter was inserted for hemodynamic monitoring. After several days, the patient was transferred to our hospital for care of congestive heart failure. Despite inotropic support, the patient’s condition continued to deteriorate, and an emergent thoracostomy was performed. The patient was intubated and ventilated.

**Case 2**

A 66-year-old male with a history of chronic obstructive pulmonary disease was admitted for management of chronic hypoxemia. A right subclavian venous catheter was inserted for hemodynamic monitoring. On the second hospital day, the patient developed hypoxemia and hypotension. A right thoracostomy was performed. The patient was intubated and ventilated.

**Conclusions**

The occurrence of contralateral pleural effusion secondary to subclavian venous catheterization is rare. However, the potential for significant morbidity and mortality underscores the importance of early recognition and appropriate intervention. The mechanism of contralateral pleural effusion in these cases is thought to be related to negative fluid balance and/or mechanical factors associated with subclavian catheterization.

**Conflict of Interest disclosure**

None.

**References**


**Contralateral Effusions from Venous Catheters (Ciment, Rotbart, Galbut)**
hydrothorax, hemotorax, phlebothrombosis,' superior vena cava syndrome,' pericardial tamponade,' aberrant catheter location,' air embolism,' and stellate ganglion block."

We describe two cases of contralateral pleural effusions secondary to transcutaneous subclavian catheterizations with attempts of radiographic contrast studies.

**Case Reports**

**Case 1**

A 79-year-old woman was admitted on July 6, 1982, with an intertrochanteric fracture of the neck portion of the left femur with avulsion of the greater and lesser trochanters, secondary to a fall. A chest x-ray film on the day of admission documented clear pulmonary fields. The patient underwent open reduction with a nail-and-plate fixation of the left femoral neck on July 12, at the time of surgery, a No 16 gauge left subclavian catheter (De Seret) was introduced for fluid management and antibiotic administration. A portable chest x-ray film documented position of this catheter in the upper superior vena cava, with no evidence of pneumothorax or hydrothorax.

Suddenly on July 13, 1982, the patient became extremely dyspneic, without cough, hemoptysis, or fever. Arterial blood gas levels showed a pH of 7.48, arterial carbon dioxide tension of 35 mm Hg, and arterial oxygen pressure of 43 mm Hg. A chest x-ray film demonstrated a very large right pleural effusion and possible superimposed pulmonary infiltrate. An emergency portable pulmonary scan performed in the intensive care unit showed minimal perfusion to the right midpulmonary zone, with normal perfusion of the left lung.

Following injection of radiographic contrast material (Conray-60) via the central venous catheter line, multiple roentgenograms showed no evidence of leakage. The patient was started on an infusion of heparin, but over the subsequent 12 hours, her condition deteriorated, hypoxemia increased, and pulmonary vessels became congested. A chest-tube thoracostomy and endotracheal intubation were accomplished. Pleural fluid obtained demonstrated the following findings: red blood cell count, 1,000/ml mm; white blood cell count, 250/ml mm, with 10 percent polymorphonuclear leukocytes, 10 percent lymphocytes, 5 percent monocytes, and 75 percent mesothelial cells; specific gravity, 1.012; glucose level, 1672 mg/100 ml; lactic dehydrogenase, 270 mg/100 ml; amylase, 5 IU/L; and protein, 1.5 g/100 ml.

The central venous catheter was removed from the left subclavian vein, and no further accumulation of fluid was noted. The chest tube was removed in 48 hours, with complete clearing of all pulmonary findings and symptoms.

**Case 2**

A 79-year-old man was admitted on Aug 31, 1981, complaining of progressive inanition, anemia, and midepigastric discomfort. The initial work-up documented an inflammatory mass of the splenic flexure associated with pancreatitis and diverticulitis. The patient was prepared for surgery with total parenteral nutrition, which required placement of successive central venous catheters. On Oct 2, 1981, the patient underwent resection of the inflammatory mass and construction of a colostomy. At the time of surgery, a No 16 gauge central venous catheter (Arro) was inserted via the left subclavian vein. A portable roentgenogram demonstrated that this catheter was at the junction of the superior vena cava and axillary vein.

The patient continued to receive hyperalimentation (Travasol and Intralipid) through the central venous catheter. His postoperative course was extremely complicated, with the onset of recurrent aspiration and progressive pneumonia. On Oct 10, 1981, the patient developed respiratory insufficiency requiring intubation and mechanical ventilation. A large right pleural effusion appeared, and a chest tube thoracostomy was necessary. Pleural fluid obtained was milky and demonstrated a glucose concentration of 1,600 mg/100 ml, lactic dehydrogenase level of 100 mg/100 ml, and triglyceride level of 1,942 mg/100 ml. There were 1,000 white blood cells per cubic millimeter, with 60 percent polymorphonuclear leukocytes, 36 percent lymphocytes, and 4 percent mesothelial cells. A radiographic contrast injection (Conray-60) showed continuity of the central venous catheter within the venous system but also demonstrated evidence of a mediastinal leak at the junction of the superior vena cava and innominate vein. A chest tube was placed, and the subclavian catheter was removed.

The patient's subsequent course during hospitalization continued downhill; he finally died two months after admission due to bilateral pneumonia and severe sepsis.

**Discussion**

The usual complications of subclavian catheterizations are well known, and it is a universal practice to introduce these aseptically, to verify blood return, and to exclude radiographically intrapleural placement and pneumothorax. The two cases presented illustrate an unsuspected complication by virtue of its late appearance (after 48 hours) and contralaterality. Although ipsilateral effusions are probably related to direct communication or penetration into the pleural space, contralateral effusions seem to occur secondarily from mediastinal leakage, as documented in case 2. This mechanism has been implicated in extrapericardial tamponade.1

It is curious that in both cases presented, the subclavian catheter entered from the left and sat at the junction of the subclavian, innominate, and aygous veins. It is speculated that angulation and combined transmitted arterial and respiratory motion contributed to the erosion at this junction. Such speculation has been suggested by others24 as causing superior vena cava perforation, leading to direct pericardial tamponade. Indeed, it is curious that both these complications tend to occur late (more than 24 hours) after insertion, at a time when clinical suspicion of iatrogenic causes would be low.

Inasmuch as patients needing such catheters usually have complicated medical problems, the etiology of late-appearing contralateral effusions may present diagnostic difficulty. A concentration of glucose in the pleural fluid approaching that of the infusate confirms leakage. Radiographic contrast studies are helpful, however, pathways of leakage may escape detection, as in case 1.

It would appear that choosing sites for insertion that reduce angulation (such as the right internal jugular vein), judicious positioning of the catheter's tip nearer the atrium, and timely discontinuation of central venous catheters would effectively reduce this late-occurring complication.

**References**

3 Friedman BA, Jurgeheit HC. Perforation of atrium by polyethylene central venous catheter. JAMA 1968; 203:1141
4 Geis PW, Johnson CF, Zajchuk R. Extrapericardial cardiac tamponade. Arch Surg 1970; 100:305
Rupture of the Left Main-stem Bronchus with a Polyvinylchloride Double-lumen Tube*

Nelson A. Burton, M.D.; Stephen M. Fall, M.D.; Thomas Lyons, M.D.; and Geoffrey M. Graeber, M.D.

Double-lumen tubes have proved to be a useful adjunct in thoracic surgery. Their use has become quite commonplace, particularly since the introduction of the softer polyvinylchloride (PVC) tubes, which are technically easier to use and may carry less risk for serious complications. Any such tube, however, can cause life-threatening complications. We present a case with such a complication related to the use of a PVC double-lumen tube and make recommendations regarding the use of these tubes to minimize the risk of serious intraoperative complications.

Selective bronchial ventilation made possible by the use of a double-lumen tube has proved to be a valuable aid to the surgeon during most thoracic operations. The advantages of these tubes are well documented. Life-threatening complications, however, including bronchial and tracheal rupture, have been reported with the Robertshaw and Carlens red rubber double-lumen tubes. The potential advantages of polyvinylchloride (PVC) double-lumen endobronchial tube have been outlined, and no cases of tracheobronchial rupture have been reported previously. We report here a case of intraoperative bronchial rupture with a PVC double-lumen tube and make recommendations regarding the management and the avoidance of such a problem.

CASE REPORT

The patient is a 72-year-old woman with a one-year history of dysphagia. She sought medical attention in June 1982, and a subsequent barium swallow revealed obstruction. Carcinoma in situ was noted on a biopsy specimen following endoscopy and dilatation. The patient was referred to the Gastrointestinal Service at Walter Reed Army Medical Center. Repeated endoscopy and biopsy demonstrated invasive squamous cell carcinoma of the distal one third of the esophagus. Further evaluation revealed no evidence of local spread or distant metastatic disease. However, a right upper lobe calcified nodule, previously noted in x-ray films from 1981, was thought to have increased in size. It was recommended the patient undergo an esophageal resection through celiotomy and right thoracotomy if there was no evidence of metastatic disease at operation.

On Sept 5, 1982, the patient was taken to surgery. After induction with sodium thiopental (Pentothal), curare, and succinylcholine for relaxation, the patient was intubated in the supine position with the PVC double-lumen tube. As is our practice, the tube stylet was withdrawn after the tip of the tube passed the vocal cords. The tube was then easily positioned. Appropriate placement was determined by auscultation, establishing that satisfactory ventilation and deflation of each lung could be carried out. When the patient was placed in a partial right lateral thoracotomy position, tube position was again confirmed by auscultation. After celiotomy, a right anterolateral thoracotomy was performed. The right upper lobe mass proved to be poorly differentiated squamous cell carcinoma, and no further resection was undertaken. To this point, arterial blood gases on one-lung anesthesia had been good with a PaO2 ranging from 155 to 270 mm Hg and the Pco2 consistently below 35 mm Hg. Shortly after the right lung was deflated, however, ventilation of the left lung was somewhat more difficult. The inflated balloon of the bronchial cuff was found to be quite tense. After evacuation of 4 to 5 ml of air from the bronchial cuff balloon, the ventilation of the left lung improved. Increasing mediastinal emphysema prompted further dissection behind the esophagus. The proximal left main-stem bronchus was found to be perforated, while the bronchial balloon was still intact. The trachea and the esophagus were then mobilized to provide adequate exposure for primary repair of the bronchial laceration, which was performed without difficulty using interrupted 5-0 prolene sutures. The repair was covered with a mediastinal based pleural flap. Chest tubes were placed, and the thoracotomy incision was closed in the usual fashion. An air leak was noted postoperatively but stopped completely by the fourth postoperative day. The patient had an uncomplicated postoperative course, with chest tubes being removed on the sixth postoperative day. Follow-up chest x-ray films and physical examinations revealed no evidence of complications. The patient was discharged on the ninth postoperative day.

COMMENT

Life-threatening complications including tracheal or bronchial rupture have been reported rarely with the older Carlens and Robertshaw tubes. These tubes, made of rather stiff red rubber latex, have low-volume, high-pressure endobronchial and endotracheal balloons. As suggested by Heiser et al in their report of a bronchial rupture, these properties of the red rubber tubes may tend to increase the risk of bronchial laceration.

It was our belief that the construction of the PVC tube with its more pliable structure, the gentle curvature of the bronchial portion of the tube, and the soft, low-pressure bronchial and tracheal balloons would carry less risk of endobronchial or endotracheal injury. While this may be true, it is obvious from this case that the potential for bronchial and perhaps tracheal rupture still exists. Following our experience, we have been made aware of one other unreported case of a bronchial injury where overinflation of the bronchial balloon was probably the etiology of rupture, since a total of 10 ml of air was instilled into the bronchial balloon. While not certain, it seems that our case also resulted from overdistention of the bronchial balloon. This was probably secondary to diffusion of nitrous oxide, however, rather than initial overdistention. As mentioned, this occurred well into the case. Initially, 2 to 3 ml of air were inflated into the bronchial balloon. However, when ventila-

---

*From the Thoracic and Cardiovascular Surgery Service and Anesthesiology Department, Walter Reed Army Medical Center, Washington, DC.
Reprint requests: Dr. Burton, Cardiothoracic Surgery Service, Walter Reed Army Medical Center, Washington, D.C. 2002