ously shown (Fig 2) demonstrating a right lower lobe A-V fistula.

**DISCUSSION**

The results in this patient show the value of the shunt measurement at high lung volume compared to FRC in the diagnosis and follow-up of A-V fistulas. The theoretical basis for the increased shunt at TLC is the increased pulmonary vascular resistance that occurs with inflation to a high lung volume. This effect is even greater in the hypoxic state. Another reason is the stretching of vessels by the surrounding expanding lung at high lung volumes (pulmonary interdependence). Most A-V fistulas are situated in subpleural areas and are subject to distension by the expanding parenchyma and would accept a greater blood flow.

In the patients described by Huseby et al, the technique was used to identify or confirm the presence of fistulas in a qualitative manner. We have utilized this technique for evaluation before and after surgical excision of A-V fistulas.

The treatment of A-V fistulas has been controversial. From his review of 63 cases at the Mayo Clinic, Dines et al recommended that surgical excision be recommended for patients with symptoms (especially hemoptysis), roentgenographic evidence of enlargement, single lesions associated with hereditary telangiectasia, and fistulas fed by a systemic arterial supply (3 of 63 in his series). Other authors have recommended that all be removed because of the danger of brain abscess. Hodgson and Kaye suggested the removal of fistulas as a frequency of usage increases. Although there are numerous variations in laparoscopic techniques, basic to all is the introduction of some form of pneumopericardium and this is the source of many complications. Although subcutaneous, preperitoneal, omental and mediastinal emphysema have been described, to our knowledge pneumopericardium has not been documented previously. This report deals with a patient who developed pneumopericardium and subcutaneous emphysema following an otherwise uncomplicated laparoscopic procedure.

**REFERENCES**


**Pneumopericardium following Laparoscopy**

R. Derek Nicholson, M.D., and Neil D. Berman, M.D.

There have been no published reports of pneumopericardium complicating laparoscopy. Following an apparently uncomplicated laparoscopy, a 35-year-old woman developed pneumopericardium associated with subcutaneous emphysema of the neck. This resolved without specific therapy and without sequela.

Laparoscopy has gained widespread acceptance in gynecologic surgery because of its efficiency, both as a diagnostic and therapeutic tool, coupled with a low morbidity. As with other innovations, potential complications become increasingly apparent as the frequency of usage increases. Although there are numerous variations in laparoscopic techniques, basic to all is the introduction of some form of pneumopericardium and this is the source of many complications. Although subcutaneous, preperitoneal, omental and mediastinal emphysema have been described, to our knowledge pneumopericardium has not been documented previously. This report deals with a patient who developed pneumopericardium and subcutaneous emphysema following an otherwise uncomplicated laparoscopic procedure.

**CASE REPORT**

A 35-year-old woman was admitted for a laparoscopic tubal coagulation. Physical examination was unremarkable. There was a soft systolic ejection murmur along the left sternal border.

Under general endotracheal anesthesia, in the dorso-lithotomy position, laparoscopy was performed using a Veress needle and insufflation with 5 liters of carbon dioxide. Good visualization was obtained and the pelvic organs were normal. A coagulation electrode was introduced through a second incision above the pubis and the fallopian tubes were coagulated. The abdomen was desufflated and the incisions closed. The total duration of anesthesia was 45 minutes.

**Pneumopericardium Following Laparoscopy**

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That evening, about six hours after the procedure, the patient complained of chest pain. There were crepitations in the subcutaneous tissue of the neck and upper anterior chest, but not in the anterior abdominal wall. On auscultation, a loud pericardial "crunch" was heard coincident with the heart rate. Blood pressure, pulse rate and venous pressure were normal. An ECG was normal.

X-ray films of the chest (Fig 1) and the soft tissues of the neck (Fig 2) documented the presence of pneumopericardium and surgical emphysema involving the soft tissues of the mediastinum and neck. There was no evidence of pneumothorax.

The patient was kept in hospital for the next three days. Vital signs were monitored and remained stable. The patient was treated with analgesics. She was discharged on the fourth postoperative day. Her examination at the time was similar to that prior to surgery except for some crepitations localized to the subcutaneous tissue above the clavicles.

**DISCUSSION**

Subcutaneous emphysema has been reported to occur in from 0.2% to 12% per 1,000 laparoscopies. The mechanism is presumed to be accidental movement of the trocar sleeve so that the insufflating ports at the tip end up in the properitoneal space. From there the gas can dissect cephalad into the mediastinum. This does not appear to have been the mechanism in this case, as the abdominal wall was never noted to be involved with emphysema. The gas may have passed through one or other of the gaps in the diaphragm and into the mediastinum and pericardium. From here, the gas tracked into the soft tissues of the neck and subsequently, because of the patient's supine position postoperatively, into the tissues of the anterior chest wall.

In the embryo, the pericardial and peritoneal cavities communicate10 and it may be that the patient had a congenital deficiency of part of the membranous portion of the diaphragm. However, congenital pericardial defects that have been described pathologically have invariably communicated with a pleural cavity.11

There was no evidence of pneumothorax in our patient. At one point during the procedure, while the peritoneal cavity was still insufflated, the patient coughed. It is conceivable that the resultant changes in intrathoracic and intra-abdominal pressures at this time forced some air through a diaphragmatic hiatus most likely around the inferior vena cava, and that this air slowly moved upward becoming manifest some several hours postoperatively.

Although tension pneumopericardium has been described as producing the syndrome of cardiac tamponade,12,13 this has always been in a setting where there has been a continued exposure to air under pressure either due to mechanical ventilation or a tension pneumothorax.12,13 In the absence of associated fluid, air in the pericardial space that is not under high pressure does not produce any evidence of hemodynamic embarrassment because of the compressibility of the air as compared to fluid.14

Thus, pneumopericardium appears to be a rare complication of laparoscopy which may result in a prolongation of the patient's hospital stay, but is unlikely to have more significant consequences.

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**FIGURE 1.** Chest x-ray film, posteroanterior projection; lowest arrow on either side indicates subdiaphragmatic air; remaining arrows point to air outlining the pericardial cavity.

**FIGURE 2.** X-ray film of neck in anterior projection showing air in the tissue planes and in the subcutaneous tissues above the left clavicle.
Bloody Pleural Effusion in a Patient with Sarcoidosis*

Paul De Vuyst, M.D.; Andre De Troyer, M.D.; and Jean-Claude Yernault, M.D., F.C.C.P.

A 50-year-old man was evaluated for pleuritic pain. Chest roentgenogram showed diffuse parenchymal infiltrates and bilateral pleural effusion that, on thoracentesis, was found to be a bloody fluid. Biopsy of para-tracheal nodes demonstrated abundant noncaseating granulomas consistent with sarcoidosis. Prednisone therapy resulted in rapid disappearance of the pleural effusion, progressive clearing of parenchymal infiltrates, and marked improvement of pulmonary function tests. Sarcoidosis should be included in the differential diagnosis of bloody pleural effusion.

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