# **Pneumopericardium**: *two case reports and a review*

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**ABSTRACT**
Pneumopericardium, and especially tension pneumopericardium, are relatively rare consequences of penetrating, blunt or iatrogenic injury. Despite its rarity and the compressibility of air, pneumopericardium can result in life threatening cardiac tamponade. We present two recent cases of this rare condition with divergent aetiologies from which lessons can be learned. The pathophysiology, diagnosis and treatment of pneumopericardium are reviewed.

## Introduction

Pneumopericardium, and especially tension pneumopericardium, are relatively rare consequences of penetrating, blunt or iatrogenic injury.[1,2] In this light, we report two instructive cases of pneumopericardium that have been brought to our attention and present a review of the topic.

## Case 1*

A 22-year-old man weighing 65 kg sustained multiple knife wounds to the left anterior hemithorax, left posterior triangle of the neck and right eye. On presentation at a rural hospital, he was haemodynamically stable and a left-sided thoracostomy tube was inserted.

The penetrating injury to the eye necessitated exploration. The patient was transferred to our institution and spent an uneventful Saturday night in the ophthalmology ward. He presented for ophthalmic surgery 16 hours after the initial assault. On arrival in the operating room, his heart sounds were normal, his blood pressure was 138/85 mmHg, his heart rate 92 per minute, and oxygen saturation measured by pulse oximetry ($SpO_2$) while breathing room air was 95% and there was no evidence of respiratory distress. A left-sided intercostal drain was in situ. The only available chest X-ray had been taken before insertion of the chest drain, which revealed a 2 mm pneumopericardium and surgical emphysema in the soft tissue of the left hemithorax. However, a review of the initial radiograph revealed no evidence for a pneumothorax. In spite of the absence of a follow-up radiograph, the anaesthetist elected to proceed with surgery.

A rapid sequence induction was followed by placement of a 7.5 mm inner diameter cuffed endotracheal tube. Intermittent positive pressure ventilation (IPPV) was commenced, delivering oxygen and air using tidal volumes of 550 ml at a rate of 10 breaths per minute. No positive end-expiratory pressure (PEEP) was administered. Ten minutes after induction of anaesthesia, $SpO_2$ had decreased from 97% to 90%. The endotracheal tube position was again confirmed by inspecting the capnogram and auscultation in both axillae. Because of greatly diminished air entry over the left hemithorax and the chest drain not 'swinging' with respiration, a pneumothorax was suspected. A new thoracostomy tube was inserted which was confirmed to 'swing' (but not bubble) with IPPV. Thereafter, 40 cm $H_2O$ continuous airway pressure was applied for 10 to 15 seconds in order to recruit any atelectatic lung. This restored $SpO_2$ to 99%. $SpO_2$ progressively decreased to 85% over the following 15 minutes and another recruitment manoeuvre was rewarded by a temporary improvement in oxygen saturation. This pattern was repeated four times.

In the light of the repeated desaturation, a mobile intraoperative chest radiograph was requested. Surgical repair of the scleral laceration was completed within 90 minutes during which time both ventilation and oxygenation became increasingly difficult. Close to completion of surgery, the lungs were being ventilated with a $FiO_2$ of 0.7, inspired tidal volumes of 450 ml at 14 breaths per minute. Peak airway pressure had increased from 18 cm $H_2O$ shortly after induction of anaesthesia, to 24 cm $H_2O$. A radial arterial cannula was placed and arterial blood gas analysis revealed pHa 7.25, $PaCO_2$ 8.3 kPa (PETCO2 7.1 kPa), $PaO_2$ 10.1 kPa, oxygen saturation 93% and haematocrit 32%. Although intraoperative arterial blood pressure always exceeded 110/70 mmHg, his heart rate progressively increased to attain 140 beats per minute by the end of surgery. The mobile chest radiograph was taken at the completion of surgery and revealed the presence of a large 2 to 3 cm pneumopericardium with a visibly diminished cardiac silhouette. Although the thoracostomy tube was in place with no evidence of a pneumothorax, the left lung field revealed loss of volume. A cardiothoracic surgeon was consulted and assurance was given that air in the pericardium is unlikely to cause haemodynamic compromise. Nonetheless, pulseless electrical activity ensued and was followed by ventricular fibrillation. In spite of effective cardiopulmonary resuscitation (CPR), as evaluated by systolic pressures of 85 to 105 mmHg and a $PECO_2$ of 3 to 5 kPa, the heart could not be restarted until transcutaneous subxiphisternal pericardiocentesis was performed. Only 20 seconds after aspirating the pneumopericardium, spontaneous cardiac rhythm and activity returned. Emergency thoracotomy was performed in the ophthalmology suite. Inspection of the lung and mediastinum revealed no obvious lacerations. In spite of blood being visible in the left main bronchus, no definitive diagnosis of bronchial laceration

**This case has been previously reported and an abbreviated version is presented here.*

could be made on bronchoscopy. Bilateral pleural and mediastinal drains were inserted. The mediastinal drain bubbled, whereas the pleural drains only swung, with IPPV.

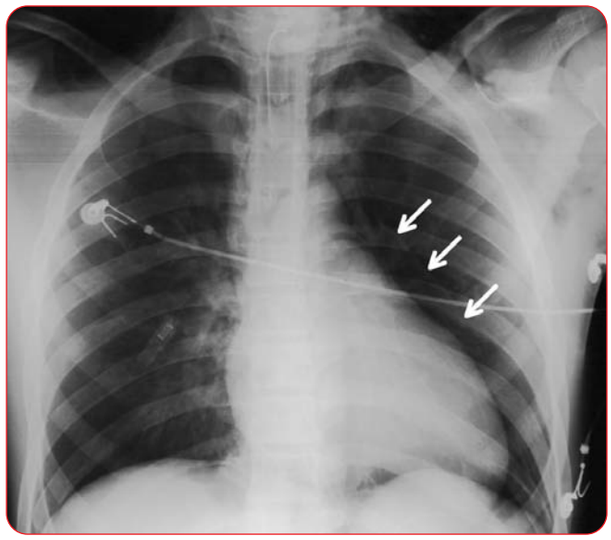
**Case 2**

A 16-year-old youth weighing 51 kg, who had been ventilated for 10 days after sustaining head injuries in a motor vehicle accident, developed progressively worse stridor. Bronchoscopy revealed the presence of a tracheal stenosis 1.5 to 2 centimetres below the vocal cords. In spite of successful dilation performed during the first bronchoscopy, the symptoms again progressively worsened. The patient was scheduled to undergo repeat examination under anaesthesia and dilation of the stenosis. Direct laryngoscopy performed two days prior to surgery in the ward revealed 70% tracheal stenosis. On arrival in theatre, respiratory rate was 14 breaths per minute and although inspiratory stridor was present and accessory muscles were being employed, the patient was supine and did not appear to be in severe respiratory distress. $SpO_2$ was 95% while breathing room air. Once preoxygenation was complete, glycopyrrolate 0.3 mg, midazolam 1 mg, alfentanil 1 mg and propofol 70 mg were administered. After the ability to ventilate the lungs using a facemask was confirmed, cisatracurium 8 mg was administered. Anaesthesia was maintained using an infusion of propofol initially at 8 and later 6 $mg.kg^{-1}.hour^{-1}$ The trachea was intubated with difficulty using an 11 French (4 mm outer diameter) "extra firm" airway exchange catheter (William Cook, Europe) and slow jet ventilation at two to three breaths per minute was commenced. The aims of adequate oxygenation rather than adequate ventilation and allowing the chest to fall after each inspiration were reiterated prior to commencement of jet ventilation. Nonetheless, over the next three minutes a progressive decrease in blood pressure from 110 to 55 mmHg systolic occurred and as bronchoscopy was about to commence, the presence of surgical emphysema in the patient's neck was noted. At this time, $SpO_2$ was still 95%. Jet ventilation was terminated immediately, the airway exchange catheter removed and slow positive pressure ventilation (four to five breaths per minute) was commenced first using a facemask and then via an endotracheal tube placed above the lesion. Hypotension was responsive to boluses of phenylephrine. Clinical examination did not reveal the presence of pneumothoraces. Subsequent direct laryngoscopy with the operating microscope revealed a tracheal stenosis with 3 to 4 mm diameter, much narrower than had been reported by preoperative direct laryngoscopy. A decision was taken to perform a tracheostomy below the lesion, which proceeded smoothly. Intraoperative chest radiograph taken at the end of surgery (Figure 1) revealed the presence of both a pneumomediastinum and pneumopericardium, but no pneumothorax. There was no haemodynamic compromise attributable to the pneumopericardium. The further intra- and postoperative course was uneventful and the patient was subsequently scheduled for tracheal resection.

**Discussion**

Pneumopericardium and tension pneumopericardium following chest trauma and positive pressure ventilation are well described, even though they are rare and poorly recognised phenomena.[3,4] Three main mechanisms can result in a pneumopericardium. The first is a macroperforation of the pericardium with communication to either the respiratory or gastrointestinal tracts.[2,5,6] The second mechanism involves a pleuropericardial connection in the presence of a pneumothorax[1,7] and the third is pulmonary volutrauma with tracking of alveolar air into the pericardium.[2,5] Of the cases of tension pneumopericardium reviewed by Cummings and colleagues,[5] 74% occurred in ventilated neonates, 14% were related to pyopneumopericardium and 11% occurred after a traumatic event.

**Figure 1**



Macroperforations of the pericardium may be caused by penetration or blunt trauma and canfollow thoracic or abdominal surgery.[2,5,6,8–11] In the first case, a penetrating injury causing a communication between a bronchus and the pericardium was the likely cause of injury. In the experience of the senior author, visualising a communication between bronchus and pericardium during bronchoscopy can be challenging. Medical conditions can also result in fistulae between the respiratory system or gastrointestinal tract, and the pericardium. These conditions include malignancies (bronchial carcinoma), and a variety of 'benign' aetiologies such as perforated duodenal and gastric ulcers, hepatic abscess and systemic and local infections.[5,12–14]

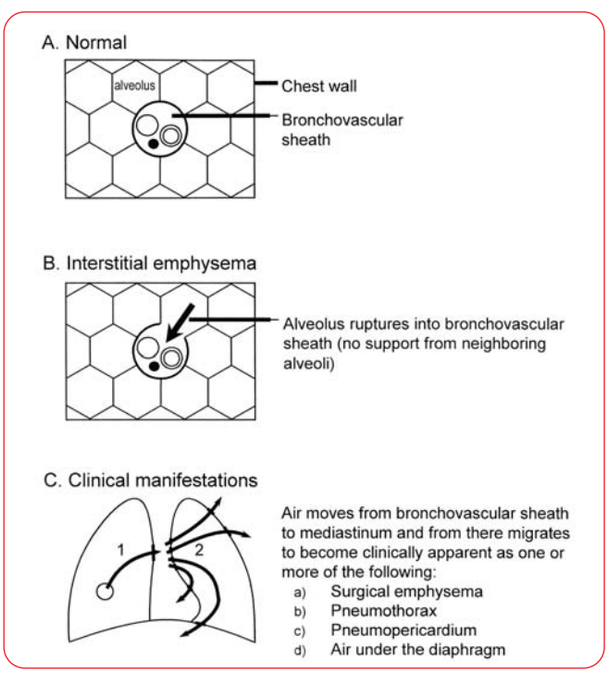
The co-existence of both a pneumothorax and also traumatic pericardial tears or congenital pleuropericardial connections can result in a pneumopericardium.[1,3,4,15–19] Congenital pathways are a potential cause of pneumopericardium during laparoscopy.[20–24] In utero, the pleuroperitoneal membranes, together with the central tendon of the diaphragm, separate the pleural, pericardial and peritoneal cavities. Should incomplete closure of these membranes occur in utero, a gap between cavities would exist and peritoneal or pleural gas could enter the pericardial sac.[25–27]

In spite of the presence of a pericardial macroperforation, clinically significant tamponade will only occur if two elements are present.[6] The first element is a source of gas under pressure and the second is a communication between the lung and the pericardial sac. Hymes and colleagues suggest that the pressure differential must exceed 14.5 cm $H_2O$ for tamponade to develop.[6] It is uncertain whether this indeed represents a critical pressure, but if a one-way communication between the lung and the pericardium does exist, IPPV may rapidly produce a life-threatening condition.[2,5,28–30] Indeed, initiation of IPPV has been associated with haemodynamic compromise in 10 of 12 patients who developed tension pneumopericardium.[1] Tamponade is unlikely if the patient is breathing spontaneously, evidenced in this case by the uneventful 16 hours between assault and initiation of IPPV.[31]

Pulmonary volutrauma during IPPV and other causes of increased intra-alveolar pressure can result in alveolar air reaching the pericardium.[32–36] This occurs with the greatest frequency in ventilated neonates and is associated with a high mortality.[5,32,37] (The mechanisms of pneumomediastinum in blunt chest trauma probably include all three aforementioned mechanisms.[36,38,39]) The process whereby volutrauma causes air to reach the mediastinum (Figure 2) has been termed the "Macklin effect".[1] The pathophysiology includes alveolar rupture followed by air dissection along bronchovascular sheaths to the mediastinum.[40] Volutrauma has been shown to cause rupture either at the terminal bronchiole at the junction with the vascular sheaths, or in the alveolus itself.[41] The mechanism of how the air arrives at the mediastinum was first determined by Macklin in experiments where he introduced hot gelatine containing minute grains of carmine into an overventilated segment of lung.[42,43] Subsequent histology showed that after alveolar rupture, air dissects along perivascular and peribronchial sheaths to the hilum of the lung. From there, air can migrate to the mediastinum. Mansfield and colleagues histologically demonstrated the existence of a potential communication between the mediastinum and the pericardium at the ostia of the pulmonary veins.[44] At this point, the pericardial collagenous tissue is not continuous with the pulmonary veins. They postulated that, in patients receiving IPPV, air could be forced along these vessels and enter the pericardial sac with relative ease.[44] Although this mechanism is frequently responsible for mediastinal emphysema in ventilated patients, tension pneumopericardium remains a rare entity.[2,5,31]

Volutrauma because of an unexpectedly narrow tracheal lumen and consequent inability to completely exhale was most likely responsible for the pneumopericardium in the second case. We and others[45–49] have successfully used an airway exchange catheter and jet ventilation for management of even relatively tight upper and lower airway stenoses. However, when using such a technique, it is **imperative** to avoid volutrauma by utilising a slow ventilatory pattern and to monitor chest wall excursion to **ensure adequate expiration** before the next inspiration is administered.[45,50–52] The aforementioned point is the most important lesson to be learned from this particular case. It must also be emphasised that adequate oxygenation and not necessarily adequate ventilation, should be the aim when using this technique. Klein and colleagues[49,53] utilised a specially manufactured double lumen catheter during resection of a stenotic tracheal lesion 4.5 cm below the glottis. While one lumen is intended to be used for jet ventilation and the other for capnography, the second lumen would in our opinion be better used for continuous monitoring of intrathoracic pressures as an attempt to prevent volutrauma.

**Figure 1**




The pathophysiological significance of pneumopericardium has evolved over time. In 1924, Oppenheimer aspirated 800 ml of pericardial effusion fluid, after which he injected 500 ml of air into the pericardium without consequence to the patient.[54] Shackleford's 1931 review states that pneumopericardium is innocuous because of the compressibility of air.[55] Subsequent work was conducted by Adcock and colleagues in a woman in whom a therapeutic pneumopericardium was initiated as part of antituberculous therapy.[56] This work contradicted earlier opinions as progressive haemodynamic compromise did indeed occur as greater amounts of air were insufflated into the pericardium. After 60 ml of air had been injected, pericardial pressures exceeded 14.5 cm $H_2O$, central venous pressure increased proportionately while cardiac output and arterial blood pressure decreased. After injecting more air, clinical signs of cardiac tamponade occurred when a certain critical pericardial pressure was exceeded, predominantly due to decreased venous return and right ventricular compression.[33,57] Adcock and colleagues and Reed and Thomas[58] described this critical pericardial pressure to be 26.6 cm $H_2O$ and 12.2 mmHg respectively. The rate of injection of air also plays a role in the development of tamponade, presumably because progressive pericardial stretch takes time to occur.[57]

The clinical signs of pneumopericardium classically include attenuation of heart sounds (usually described as distant, muffled or absent) as occurs in pericardial effusions.[5,59] However, auscultation may also reveal other characteristic sounds. Bricheteau described a sound similar to that made by the floats of the millwheel as they strike the water.[57] He termed this sloshing murmur in his patient with pyopneumopericardium as a "Bruit de Moulin".[5,57] On occasion, either a friction rub or Hamman's sign (a pericardial crunching sound) may be audible.[57] Percussion may reveal "shifting precordial tympany" in which the precordial hyperresonance shifts as the patient changes position.[57] These clinical signs may be difficult to elicit in the emergency situation.[1,57] The clinical presentation of tension pneumopericardium is with the classic signs of cardiac tamponade. These include elevated central venous pressure, hypotension, tachycardia, pulsus paradoxus, and diminished heart sounds on auscultation.[2,5,57,60,61] In spite of tamponade typically causing a tachycardia, bradycardia has been observed with pneumopericardium.[5,29,56,57] Electrocardiograph may reveal tachycardia or bradycardia, a global low voltage or changes similar to those observed in pericarditis.[2,5,29,62]

Chest radiography typically shows a small heart, partially or completely surrounded by air, which is contained within a sharply defined halo of pericardium (the halo sign).[2,63,64] Furthermore, a sudden decrease of more than 2 centimetres in the cardiac shadow compared to the initial chest radiograph or a 33% reduction in cardiac size[64] should alert the clinician to

the possibility of a tension pneumomediastinum. Mirvis and colleagues have called this the "small heart sign".[2]

The radiographic signs of pneumopericardium and pneumomediastinum can be similar.[5,59,65] In pneumomediastinum, air is visible radiographically as streaks that are not limited by the pericardial boundaries and that stretch into the superior mediastinum. The air does not surround the heart or shift when the patient is put in the lateral decubitus position.[2,28,31] However, in the presence of a pneumopericardium, the pericardium confines the spread of air to below the aortic notch.[59,63] Signs that are pathognomonic of pneumopericardium are the "transverse band of air" sign on the frontal radiograph that represents air in the transverse sinus of the pericardium and the "triangle of air" sign visible on the lateral chest radiograph.[66] The diagnosis has also been facilitated by CT scan[67] and transthoracic or transoesophageal echocardiography, visualising signs of pericardial tamponade or the presence of pericardial air.[29,63,68–70]

The immediate treatment of tension pneumopericardium is decompression of the pericardial air by insertion of a large-bore needle or intravenous catheter. This treatment has on occasion, as in this case, produced dramatic return of spontaneous circulation.[30,71] A small tube (e.g. 24 French gauge chest tube or pigtail cardiac catheter)[72] may be subsequently inserted into the pericardium via a subxiphoid approach.[6] These drains may block and consideration should be given to the creation of a pericardial window.[73] A potential problem with pericardial decompression may occur in patients with diminished lung compliance undergoing IPPV, with preferential movement of air from the lungs through the pericardial drain. This may cause a broncho-pericardial-cutaneous fistula that may make ventilation difficult.

How would one manage a patient presenting with a preoperative pneumopericardium?[74] If the pneumopericardium was substantial and haemodynamically significant, pericardiocentesis would obviously need to be performed immediately, before anaesthesia or IPPV is commenced. However, what should an anaesthetist do if the preoperative chest radiograph indicates a small, clinically insignificant pneumopericardium? The relevance of such a radiographic finding is emphasised by two unrelated case series,[1,5] both reporting a 37% incidence of tension pneumopericardium if IPPV was commenced in the presence of pericardial air. There are four important points that need to be made when considering anaesthesia for such a patient:

1. Attempts should be made to avoid aggravating the pneumopericardium. We would suggest that, if possible, it may be prudent to avoid general anaesthesia. If general anaesthesia is unavoidable, inspired tidal volumes should be limited and lung recruitment manoeuvres must definitively be avoided.
2. The anaesthetist should be vigilant for both the common and non-specific signs (hypoxia, tachycardia) that may indicate that a tension pneumopericardium is developing. There should be immediate intraoperative access to chest roentgenography for the purposes of diagnosis.
3. It is imperative that the ability to manage a pneumopericardium be available. This involves equipment, expertise and willingness to perform pericardiocentesis.
4. The "H's" and "T's" need to be considered when patients are unresponsive to CPR.[75]

In the first case, the tension pneumopericardium presented unusually with hypoxaemia, rather than with haemodynamic compromise. The chest radiograph revealed that the left lung demonstrated a low lung volume. Very large pericardial effusions have been reported to compress the left lower lobe bronchus and result in left lower lobe collapse.[76,77] While we hypothesise that the pneumopericardium caused compression of the left lower lobe, the possibility of an (undiagnosed) fistula between the left lower lobe and the pericardium causing loss of volume cannot be excluded. Furthermore, the tension pneumopericardium would have decreased cardiac output significantly, increased tissue oxygen extraction and resulted in a low mixed venous oxygenation. The combination of a low mixed venous oxygen content and low ventilation perfusion units or shunt will aggravate hypoxaemia.[78–80] While recruitment manoeuvres should reduce atelectasis,[81] it will reoccur within minutes when lung recruitment is performed in the presence of a high inspired oxygen fraction. This explains the rapid and repeated reoccurrence of arterial hypoxia after recruitment.[82,83] Recruitment manoeuvres could also have aggravated arterial hypoxaemia by worsening the pneumopericardium and further decreasing cardiac output.[84–86]

These case reports emphasise a number of points:

1. A tension pneumopericardium can cause as significant a haemodynamic compromise as pericardial fluid.
2. Pneumopericardium is usually innocuous during spontaneous respiration but can become rapidly life-threatening during IPPV in the presence of even a small communication between the respiratory tract and pericardial sac. If a non-tension pneumopericardium is diagnosed before or during IPPV, a low tolerance should be maintained for performing pericardial drainage if haemodynamic compromise develops.
3. Young subjects with good cardiovascular and autonomic reserves can compensate for a considerable period before haemodynamic compromise occurs as a result of pericardial tamponade.
4. In the absence of a macroscopic connection between the respiratory tract and pericardial sac, the mechanism whereby a pneumopericardium develops is not obvious and is reviewed.
5. While arterial hypoxaemia is not usually a feature of a low cardiac output state, it can occur if both low mixed venous oxygen content and a shunt co-exist.
6. We report two cases complicated by pneumopericardium that have been brought to our notice. While the reporting of complications is not popular, this represents an opportunity to advance the safety of anaesthesia practice.[87] **SAJAA**

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