

Pneumothorax and pneumomediastinum

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Pneumothorax (PTX) and pneumomediastinum (PTM) occur uncommonly in association with athletic participation. Although they are rare, when they occur they can be life-threatening, requiring immediate diagnosis and treatment. These injuries also present difficult return-to-play (RTP) decisions for the sports medicine physician. There are sparse data to help determine the incidence of these injuries in sport, as well as their optimal treatment. Although most sports physicians have seen these injuries, not many have seen enough to publish a large series discussing optimal management or make RTP recommendations.

Definitions

PTX is defined as air that has leaked into the pleural space, either spontaneously or as a result of traumatic tears in the pleura following chest injury or iatrogenic/surgical procedures. Treatment of PTX entails removing air from the pleural space, re-expanding the underlying lung, and preventing recurrence [1]. PTX can be categorized as either spontaneous, traumatic, or iatrogenic. Spontaneous PTX can be further subdivided into primary or secondary depending on whether underlying lung disease is absent or present, respectively. Traumatic PTX can be further subdivided into penetrating or nonpenetrating trauma.

PTM is air that has leaked into the mediastinum, with mechanisms similar to that seen with PTX. PTM is often treated conservatively with careful observation. Rarely, intervention is necessary. PTM is often seen in conjunction with PTX, though symptoms may help differentiate the two. The majority of this article focuses on PTX.

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Primary spontaneous PTX occurs without underlying pulmonary disease. Individuals are often thin, tall, and may have a history of smoking or substance use [2,3]. The latter includes heroine, ecstasy, marijuana, speed, and cocaine. It has also been theorized that primary spontaneous PTX can occur as a result of the rupture of subpleural blebs (which form by an unclear mechanism), which is caused by the increased intrathoracic pressure against a closed glottis (Valsalva maneuver) [4–6]. Secondary spontaneous PTX often occurs in association with underlying pulmonary disease (most commonly chronic obstructive pulmonary disease), and individuals are often older than 35 years old. Secondary spontaneous PTX has also been reported in individuals with Marfan's syndrome, where connective tissue defects due to faulty gene coding cause an increased incidence of apical pleural blebs, which then rupture [6].

Roughly 20,000 new cases of spontaneous PTX are reported each year [7], though the majority are not associated with exercise [8]. Activity has been reported to be related to PTX in less than 10% of spontaneous PTX cases [2]. Young men are at risk, with those aged 20 to 40 years at greatest risk [8–10], and an overall male-to-female predominance of 6:1 [4]. There does not seem to be an increased incidence of spontaneous pulmonary air leaks at high altitude [11], though the same may not be true during scuba diving or other compressed air diving activities [12,13]. Barotraumas in the latter situation may result in pulmonary air leaks that occur when the individual holds their breath during the ascent, an application of Boyle's Law. A PTX that occurs underwater during an ascent may rapidly progress to a tension PTX, more so than one on land, and thus a history of spontaneous air leak is considered an absolute contraindication to scuba diving [12,14].

Curtin and colleagues [15] reported a case of spontaneous PTX in a basketball player who presented with acute onset of left-sided chest pain. The possibilities of straining with a bowel movement or air travel the day before were considered, but otherwise there was no history of trauma. The pain increased during a pregame warm-up, and the examination was remarkable with a heart rate of 60, respiratory rate of 12, blood pressure of 120/70, and decreased breath sounds in the left upper lung field. A chest radiograph confirmed a large left-sided PTX. The athlete was treated with chest tube placement for 2 days, was discharged from the hospital 3 days later, and was allowed to return to full play at 3 weeks (see Figs. 1 and 2).

Traumatic PTX can be associated with penetrating or nonpenetrating trauma, with associated fractures being common. Rib fractures are common, and certain rib segments that are associated with a higher likelihood of associated injuries and complications include the first four ribs, the last two ribs, fracture of multiple ribs, and flail segments [16]. Because rib fractures in children require a greater impact force to occur, these injuries are often associated with a higher morbidity and mortality [17]. The reported incidence of PTX in association with scapular fractures is between 11% and 38% [18,19], although in one study it occurred in 16 of 30 patients with scapular fracture [20]. In general, when a rib or scapular fracture does occur in an athlete, careful observation and consideration for the development of PTX is important.

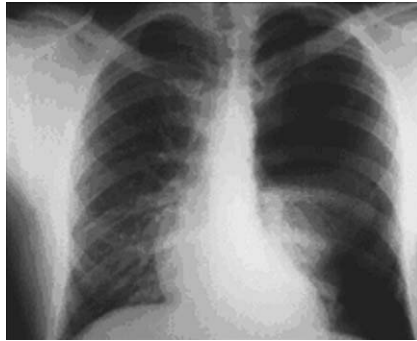


Fig. 1. A chest radiograph of a 22-year-old male college basketball player shows a large pneumothorax on the left side. The patient did not have any rib fractures or evidence of tumor or infection. (From Curtin SM, Tucker AM, Gens DR. Pneumothorax in sports: issues in recognition and follow-up care. *Phys Sportsmed* 2000;28:25; with permission.)

Iatrogenic PTX occurs as a result of various medical procedures, including thoracentesis, central line placement, subacromial injections, as well as other more invasive procedures. Tension PTX is an uncommon complication of PTX and is defined as a progressive collection of air in the pleural space [21]. The development of a tension PTX can be slow or quick depending on the degree of lung injury and the underlying health of the patient. The deterioration that occurs in tension PTX is a multifactorial process that includes hypoxemia, compensatory mechanisms, and mechanical obstruction. Hypotension occurs late, just before cardiorespiratory collapse. Additional symptoms include progressive hypoxemia, tachycardia, and respiratory distress. Tension PTX is a life-threatening emergency that must be considered in the athlete who presents with respiratory distress. **Box 1** classifies the various forms of PTX classification and barotraumas.

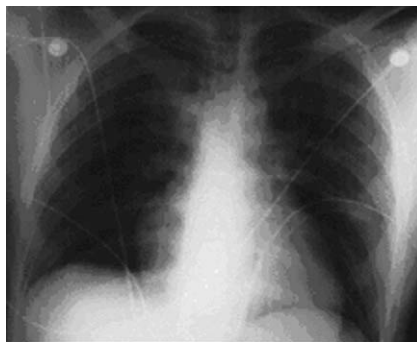


Fig. 2. A chest radiograph of the same patient in Fig. 1 taken after placement of a chest tube shows that the lung had re-expanded. The patient recovered after 2 days and was released on hospital day 3. He returned to play 3 weeks later without difficulty. (From Curtin SM, Tucker AM, Gens DR. Pneumothorax in sports: issues in recognition and follow-up care. *Phys Sportsmed* 2000;28:25; with permission.)

Box 1. Classification of PTX and barotrauma

Spontaneous PTX

- Primary (no lung disease, young, male, slender, rupture of blebs)

- Secondary to underlying lung disease (chronic obstructive pulmonary disease, asthma, cystic fibrosis, pneumocystis pneumonia, sarcoid, neoplasm, Marfan's syndrome)

- Catamenial PTX (associated with menstruation; 3rd to 4th decade of life; often on right side)

- Drug addicts (inhalation [eg, cocaine, tetrahydrocannabinol] or intravenous drug use into subclavian, internal jugular vein)

AIDS

- Familial spontaneous PTX

- Traumatic PTX (blunt or penetrating trauma; open, closed, tension and hemo-PTX)

Iatrogenic PTX

- Barotrauma and PTX associated with mechanical ventilation (often during positive pressure ventilation, large tidal volume, high positive-end expiratory pressure, high transpulmonary pressure)

Accidental

- During diagnostic procedures (central line, biopsy, aspiration)

- During therapeutic procedures (cardiopulmonary resuscitation, pulmonary function tests, nasogastric tube placement tube)

Special situations

- PTX ex vacuo

- Sports-related PTX

- Barotrauma unrelated to mechanical ventilation

- Postoperative air space

- Barotrauma in airplane passengers, pilots, and divers

- Spontaneous PTX following contralateral pneumonectomy

- Spontaneous PTX in pregnancy

Adapted from Jantz MA, Pierson DJ. Pneumothorax and barotraumas. Clin Chest Med 1994;15:75–91, with permission; and Karnik AM. Management of pneumothorax and barotraumas: current concepts. Compr Ther 2001;27:311–21, with permission.

The literature regarding PTX and PTM in the athletic realm is sparse. Although several case reports of PTX in sport have been reported, a large series of these injuries in athletes is lacking. Spontaneous PTX has been reported in joggers [22], weight lifters [5,23], scuba divers [24], and basketball players [15]. Traumatic PTX has been reported in several sports as well, including football [15,25,26], hockey [25], diving [27], boxing [28], soccer [29], weight lifting [30], and fencing [31]. Traumatic PTX in sport in association with various fractures have also been reported, including rib fractures [16], scapular fractures [18,32], and clavicle fractures [33]. PTM has also been reported during sporting activities; these injuries are most often associated with trauma, fractures, and PTX [9,25,34,35]. Spontaneous PTM has been observed in sport, but there is only one report of this [19].

The largest series of PTX in sport was reported in 1999 by Kizer [36], in which 20 cases of confirmed PTX over a 5-year span were retrospectively reviewed. All of these occurred during winter sports participation and were presented to a community hospital in the Lake Tahoe area. In 19 cases (95%), PTX was due to blunt chest trauma. Nine different sports were involved, though 55% of the cases occurred during skiing or snow boarding, most likely a result of the patient population in this community. Of the 16 patients who were alert and responsive in the emergency department, 100% presented with localized or generalized chest pain, 75% had dyspnea or shortness of breath, 69% had diminished breath sounds unilaterally, 31% had subcutaneous emphysema, and 19% had palpable crepitation at the site of their chest pain. Three patients (15%) in the series died; 53% [9] of the surviving patients required hospitalization for chest tube and treatment of their other injuries. These patients had sustained their injuries due to trauma, either from a fall or collision; the patients who died had sustained multiple trauma in which the PTX was not believed to be the cause of death. Four patients in this series were seen more than 24 hours after their injury; three others were also delayed presentations, but not as long as 24 hours. All in all, 35% delayed their visit to the emergency department for several hours. The authors surmised that despite the immediate onset of symptoms, athletes downplay their symptoms.

Partridge and colleagues [25] reported on two cases of PTX and 1 case of PTM in association with sport activity, all of which were slightly atypical in their presentation in both signs and symptoms. The delay in presentation in these cases ranged from 3 hours to the next day. Ciocca [30] reported on a dramatic case in which the presentation of PTX was delayed for 3 days. This case involved a weight lifter who developed symptoms of dyspnea, right-sided chest pain, and feeling as if his “wind had been knocked out” after “bouncing” a bar of his chest during a bench press. This athlete continued to lift weights, continued with activities of daily life, and felt his symptoms would go away, and finally sought medical attention after 3 days because the symptoms had not cleared. Chest radiographs confirmed a large right-sided PTX with total collapse of his right lung (Fig. 3). The athlete required emergency chest thoracostomy for 24 hours, with complete recovery and RTP at 1 month.

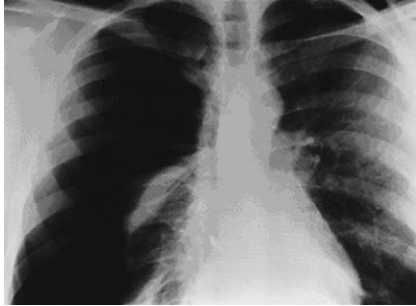


Fig. 3. A posteroanterior chest radiograph of a 27-year-old weight lifter revealed a large right-sided pneumothorax with collapse of the lung. The diagnosis is confirmed by an absence of lung markings peripheral to the visceral pleural line. (From Ciocca M. Pneumothorax in a weight lifter. *Phys Sportsmed* 2000;28; with permission.)

Ferro and McKeag [19] reported on a case of PTM during breath-holding exercises in a 19-year-old swimmer. The athlete reported mild right-sided neck pain during the practice, and was given ibuprofen by the athletic trainer. Two days later, after the first swim meet, the athlete reported increased pain, shortness of breath, and difficulty swallowing and was subsequently referred to the emergency department. Bloodwork, radiographs, and a barium swallow were all performed and read as normal, though on the following day the radiologist confirmed PTM (Figs. 4, 5). The conclusion from reviewing these reported cases

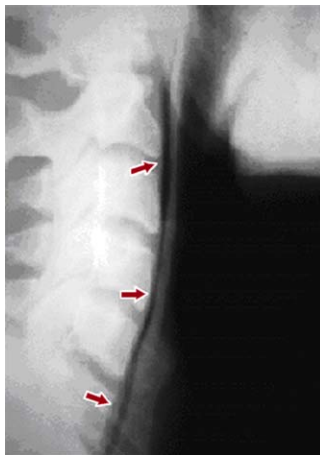


Fig. 4. A lateral cervical-spine radiograph in a 19-year-old male college swimmer reveals air in the deep cervical tissues behind the trachea and larynx (arrows) posterior to the nasopharynx. (From Ferro RT, McKeag DB. Neck pain and dyspnea in a swimmer. *Phys Sports Med* 1999;27; with permission.)

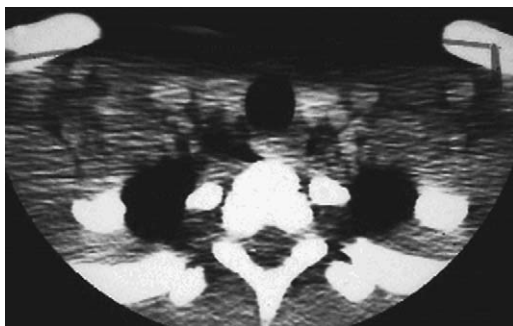


Fig. 5. A CT scan of the same patient in Fig. 4 obtained 4 days after onset of the patient's symptoms revealed only a small amount of residual air in the deep cervical tissues on the right side, posterolateral to the trachea at the level of the lung apices, and confirmed the absence of pneumothorax or subpleural blebs. (From Ferro RT, McKeag DB. Neck pain and dyspnea in a swimmer. *Phys Sports Med* 1999;27; with permission.)

of PTX and PTM in sport is that a high index of suspicion should be maintained in athletes who have a history of blunt trauma to the chest.

Evaluation

The classic presentation of PTX is dyspnea and pleuritic chest pain. These symptoms are generally present in 80% to 90% of patients, though up to 10% of patients will be asymptomatic [3]. The chest pain is usually ipsilateral, and may radiate to the shoulder, neck, and into the back. It is often pleuritic, associated with dyspnea on exertion, or a dry cough [15]. On physical examination, tachycardia, tachypnea, hyperresonance to percussion, diminished breath and voice sounds (Laennec's signs), and asymmetrical chest wall expansion may be present. If there is evidence for tracheal deviation away from the PTX, distended neck veins, hypotension, respiratory distress, or cyanosis, then tension PTX must be considered and treated emergently. With PTX, an electrocardiogram will often demonstrate a right axis shift, decreased QRS amplitude, and precordial T wave inversion.

PTM presents slightly differently, with sharp chest pain and subcutaneous emphysema over the supraclavicular area and anterior neck. There may be a characteristic "crunching sound" on auscultation over the pericardium synchronous with the heartbeat (Hamman's crunch). In addition, the athlete may complain of dysphagia or dysphonia [15].

If an athlete is evaluated and PTX or PTM is considered, the initial evaluation should include vital sign assessment and auscultation in a quiet setting. The latter may require relocation to a more conducive site, because it is often difficult to perform on a sideline near a loud crowd. If the athlete is hemodynamically unstable and findings are consistent with tension PTX, then emergent needle aspiration with an 18-gauge needle between the second and third rib followed by

thoracostomy should be performed. Oxygen should be supplied and an intravenous line should be placed, with transportation to an emergency facility secured.

The differential diagnosis of blunt trauma to the chest includes PTX (with or without complication of fracture), PTM, rib contusion, costochondral separations, muscle strains, and fracture. Confirmation of PTX is determined by a posterior–anterior radiograph that demonstrates an absence of lung markings outside of the pleural line. Small PTX may require expiratory films in a lateral decubitus position [4] to optimize visualization. The amount of lung involvement can be estimated by the following equation: $PTX\% = 1 - (\text{lung diameter}^3 / \text{hemithorax}^3) \times 100\%$ (Figs. 4, 5) [6]. CT is particularly useful for PTM when these cannot be visualized with radiography.

In 2002, Holmes and colleagues [37] published a clinical prediction protocol to identify children who have thoracic injuries after sustaining blunt torso trauma. This involved a prospective evaluation of 986 children who were seen in the emergency department after blunt trauma and who then underwent chest radiography. In this series, 80 patients had thoracic injuries, including pulmonary contusion (71%), hemothorax (35%), PTX (25%), PTM (8%), tracheal-bronchial disruption (3%), and aortic injury (3%), along with hemopericardium, cardiac contusion, rib fracture, sternal fracture, and diaphragm injury. They then performed a multiple logistic regression and recursive partitioning analysis and found that predictors of thoracic injuries included: low systolic blood pressure, elevated age-adjusted respiratory rate, abnormal thorax exam, abnormal chest auscultation, femur fracture, and a Glasgow coma scale of less than 15. Ninety-eight percent of patients who had thoracic injury had at least one of these predictive factors. It is important to realize that this series was not specific to athletes and involved primarily high-velocity injuries such as motor vehicle accidents, automobile–pedestrian, automobile–bicycle, and bicycle accidents, as well as assault, crush injuries, and abuse injuries. Although this limits the application of these predictors to injuries in athletes, they are also the only information that has been published with regard to thoracic injuries in children.

Treatment

The treatment of PTX depends on how large the PTX is, as well as the clinical stability of the patient. In 1999, the American College of Chest Physicians published guidelines for the management of spontaneous PTX [38]. If the patient is clinically stable and the PTX is less than 15% to 20%, then close observation in the emergency department for 3 to 6 hours with monitoring of vital signs and pulmonary symptoms is adequate, as long as a chest radiograph is obtained before discharge and after an additional 12 to 48 hours. If the PTX is greater than 15% to 20%, and the patient is clinically stable, then the patient should be admitted to the hospital, and a small bore chest tube should be inserted. If there is a persistent leak in primary PTX, then thoracoscopy is preferred over pleurodesis, and if the individual has a second PTX, then thoracoscopy is also a consideration.

Small tubes can be used for low risk patients, but larger tubes are preferred for patients at high-risk air leaks. As a general rule of thumb, air from the pleural space is absorbed at a rate of 1.25% per day [39]. Supplemental oxygen, which increases the pleural absorption of air, can accelerate resolution threefold [6], and thus should be used for this reason.

Treatment of tension PTX has been discussed previously. PTM will often resolve spontaneously without intervention or complication. Evaluation of the airways and esophagus is essential to rule out disruptions, and this often involves bronchoscopy and esophagoscopy or barium swallow, respectively.

Recurrence

The recurrence of primary spontaneous PTX is variable, and somewhat dependent on the treatment given. After chest tube placement, the recurrence rate has been reported as being between 10% and 21% [40]. After simple aspiration, the recurrence rate has been reported as being between 20% and 50% [39]. One report, however, found that over a 10-year course, the recurrence rate after spontaneous PTX in 124 adults was 27%, no matter the treatment received [41]. When recurrence does occur, it most often occurs on the same side, though not always. The recurrence rate increases with each recurrence [15], and is felt to be at its highest likelihood within the first few months [7].

The recurrence rate after traumatic PTX is not felt to be any different than the risk for the initial incident rate [30], although in one case report, recurrent traumatic PTX occurred in a football player who was allowed to return to play 1 week after his initial PTX [15]. If recurrence occurs, pleurodesis, thoractomy, videothoracoscopy, and median sternotomy are all considerations [7]. Thoracoscopic resection of subpleural blebs is recommended after the third spontaneous PTX or in a patient in whom tube thoracostomy is unsuccessful [15].

Return to play

There are little data to guide RTP decisions after PTX and PTM in sport. Although there are several case reports, there are no large long-term follow-up series. However, despite these limitations, there are some conclusions that can be drawn from looking at the case reports. Curtin and colleagues [15] described a spontaneous PTX in a basketball player who required chest tube placement and returned to play after 3 weeks. In the same publication, the authors described a traumatic PTX in association with a PTM in a football player who was treated with hospitalization and observation. The PTM resolved in 2 days, the PTX resolved in 5 days, and the athlete returned to play after 2 weeks. Volk and colleagues [3] described a traumatic PTX in a football player who returned to conditioning after 4 weeks and full play after 10 weeks. Levy and colleagues [42] described three cases of traumatic PTX, all of whom were treated with chest tube

placement, conditioning within 2 days of tube removal, contact drills once dyspnea resolved, and full play within 2 weeks.

In 1999, Curtin and colleagues [15] proposed RTP guidelines for PTX in athletes. Previously, it had been recommended that, given the high risk of recurrence, athletes who had sustained spontaneous PTX should be restricted to sedentary, nonstrenuous activity unless treated with thoracotomy [43]. Others felt that activity should only be restricted if the spontaneous PTX was exertional [44], which is not commonly the case. Curtin and colleagues concluded that for spontaneous PTX, the recommendation is that athletes should be allowed to return to all activities but should be counseled on the high risk of recurrence. RTP can usually occur 3 to 4 weeks after resolution of PTX, and if PTX recurs, then a corrective procedure should be performed. For traumatic PTX, there is no data to suggest that there is an increased risk for recurrence. Given this, there is no reason to restrict RTP in athletes who have sustained traumatic PTX after the PTX has resolved. In fact, it seems plausible that with scarring, the risk for recurrence after traumatic PTX may be decreased. Based on available data, RTP after traumatic PTX can usually occur 3 to 4 weeks after resolution.

Air travel

There are no clear recommendations with regard to air travel. If an athlete has acute, unresolved PTX, air travel should be avoided. For the military, the recommendations are that air travel be restricted for 6 to 9 months. This is due to the significant changes in intrathoracic pressures, as well as the responsibilities of a pilot [8,45]. For commercial travelers, a 1 to 2 month delay is recommended [15], although this is quite controversial [10]. Many argue that, especially after traumatic PTX, there is no arguable increased risk, and that again the scarring may decrease the risk for PTX, thus making travel possible once the PTX resolves.

Take-home messages

Whenever an athlete sustains blunt chest trauma, and reports pleuritic chest pain, shortness of breath, or dyspnea on exertion, PTX or PTM should be considered. The trauma does not have to be significant, and it does not have to involve fracture. When a PTX does occur, given that traumatic PTX has a more favorable outcome and much lower recurrence rate, it is useful to look for the possibility that trauma was indeed involved. Remember that the presentation of PTX or PTM may be delayed, with an athlete presenting hours to even days later. It is also important to ask about the use of illicit drugs (eg, heroine, marijuana, ecstasy, heroine, speed) and cigarettes, because these substances increase the risk of spontaneous PTX. In athletes, a higher index of suspicion should be maintained, because their fitness may mask serious intrathoracic injury [11]

and athletes often minimize their symptoms [36]. The treatment of PTX is based on the size and type of PTX, as well as the clinical symptoms that are present. Finally, RTP decisions should be individualized.

Questions for further research

There is a paucity of information regarding the natural history of PTX and PTM in sport. It is difficult to discern the exact incidence of both spontaneous and traumatic PTX in the realm of sport, as well as the outcome after various treatments. Are there factors that increase the risk of occurrence and recurrence? Are there other clinical decision tools that can be used to help make treatment and RTP decisions in athletes? More information is needed to provide reasonable recommendations for travel after PTX and PTM.

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