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Management of pneumothorax using thoracostomy and autologous blood patch pleurodesis in a dog and a cat

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ABSTRACT: This case series describes the management of two critically ill animals with pneumothorax of different etiologies. The first case involves a five-year-old male mixed-breed dog that sustained multiple thoracic bites. The dog was presented with severe respiratory distress, subcutaneous emphysema, pale mucous membranes, tachycardia, and crackling respiratory sounds. Thoracic radiographs revealed pneumothorax, pneumomediastinum, atelectasis, and flail chest due to rib fractures. Initial stabilization included oxygen therapy, thoracentesis, intravenous fluids, analgesics, and antibiotics. Due to persistent air accumulation, autologous blood patch pleurodesis (ABPP) was performed, resulting in initial clinical improvement. However, respiratory deterioration necessitated the placement of a thoracostomy tube with continuous suction. Forty-eight hours later pneumothorax was resolved, and the dog was discharged. Two years post-discharge, the dog remains asymptomatic. The second case involves a 10-month-old female cat with chronic respiratory distress due to trauma. Clinical examination revealed abdominal breathing, paradoxical movement, and dysmorphia of the thoracic wall. Radiographs were indicative of a diaphragmatic hernia. Surgical repair via midline laparotomy was performed, and thoracentesis was conducted to manage the pneumothorax. However, due to severe postoperative dyspnea and persistent air accumulation in the thorax, repeated thoracenteses, thoracostomy tube placement, and ultimately ABPP were deemed necessary. Suspicion of a ruptured pulmonary bulla led to an exploratory thoracotomy, during which a pulmonary defect was identified and resected. Unfortunately, the cat succumbed due to presumed prolonged hypoxia during surgery. These cases highlight the complexity of managing pneumothorax in small animals. The series underscores the need for a combination of classic pneumothorax management techniques with more recently described options, such as ABPP. It also emphasizes the importance of advanced diagnostic imaging in managing such patients and the variable prognoses as well.

Keyword: pneumothorax; autologous blood patch; pleurodesis; thoracostomy; trauma

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INTRODUCTION

Pneumothorax is a condition defined by the accumulation of air in the pleural space, which can lead to notable alterations in cardiopulmonary physiology (Bennett et al., 1989; Kramek & Caywood, 1987; Pawloski & Broaddus, 2010). In terms of etiology, it is broadly classified as either spontaneous or traumatic. Traumatic pneumothorax may occur as a result of blunt or penetrating trauma, iatrogenic causes (i.e. thoracentesis, barotrauma), or even as a postoperative complication following lobectomy or diaphragmatic herniorrhaphy, particularly in chronic cases (Pawloski & Broaddus, 2010; Sandoval et al., 2024; Simpson et al., 2009). Spontaneous pneumothorax is often primary, resulting from the rupture of pulmonary bullae or blebs, or may be secondary, associated with neoplasia, thromboembolism, parasitic infections, or other forms of pneumonia (Gilday et al., 2021; Pawloski & Broaddus, 2010). Further classification includes open and closed types, determined by the presence of thoracic wall defects and direct communication between the pleural space and the external environment (Kramek & Caywood, 1987; Pawloski & Broaddus, 2010). A particularly severe form, tension pneumothorax, occurs when the pleural defect functions as a one-way valve, allowing influx of atmospheric air into the pleural space during inspiration but preventing removal during expiration (Hardie, 2023). If untreated, tension pneumothorax can precipitate respiratory and cardiovascular collapse due to progressive air accumulation and continuous compression of intrathoracic structures, making prompt recognition and intervention critical.

Clinical signs vary depending on the type of pneumothorax, degree of air accumulation, and the underlying etiology (e.g. trauma) (Hardie, 2023; Pawloski & Broaddus, 2010). Common respiratory symptoms include rapid shallow breathing, dyspnea, neck extension, and cyanosis. Non-specific systemic manifestations such as anorexia, lethargy, and vomiting may also occur. On thoracic auscultation, muffled heart and respiratory sounds can be indicative of pleural disease. A definitive diagnosis is established through thoracentesis, thoracic radiographs, and/or ultrasound (Boysen, 2021; Gilday et al., 2021; Pawloski & Broaddus, 2010). Computed Tomography (CT) has higher sensitivity than radiographs for detecting minimal volumes of air and bullae (Au et al., 2006), although this may depend on the size of the bulla (Reetz et al., 2013). However, its use is less common due to limited availability, longer procedural time, the need for patient sedation or general anesthesia, and its higher cost.

Management of pneumothorax may be challenging for the veterinarian. In mild, clinically stable cases, the air within the pleural space may be absorbed over a few days, requiring no further intervention except oxygen support and cage rest. However, in cases where patients present with severe respiratory distress due to increased air accumulation, decompression of the pleural cavity is often mandatory. Thoracentesis serves as a valuable procedure for diagnosis, initial stabilization as well as ongoing management. In cases of rapid air accumulation, placement of a thoracostomy tube (TT) may be required to facilitate continuous or intermittent suction (Gilday et al., 2021; Hardie, 2023; Lyons, 2022). When air accumulation cannot be resolved, surgical intervention is typically indicated after 3 to 5 days (Dickson et al., 2021; Hardie, 2023; Lyons, 2022; Mooney et al., 2012). Spontaneous pneumothorax treatment through thoracoscopy is widely used in humans (MacDuff et al., 2010; Shaikhrezai et al., 2011) and has also been successfully described in dogs (Case et al., 2015; Puerto et al., 2002) and cats (Radlinsky, 2014). Autologous blood patch pleurodesis (ABPP) has recently been described in both human and veterinary literature as a less invasive technique for successful management of pneumothorax (Merbl et al., 2010; Moses et al., 2023; Oppenheimer et al., 2014; Satmary et al., 2023; Shakir et al., 2023; Théron et al., 2021; Wong & Mooney, 2020). It involves infusing the patient's whole blood into the pleural space to achieve pleurodesis between the parietal and visceral pleura, effectively sealing defects that allow air influx into the cavity.

This case series aims to present two pneumothorax cases with distinct etiologies and to discuss conventional as well as alternative management approaches, such as ABPP.

CASE HISTORY

CASE 1 (C1)

A 5-year-old, 7 kg, intact male mixed-breed dog, was referred with multiple thoracic wounds caused by dog bites. Initial management included cleaning and suturing of the external wounds by the referring veterinarian.

Upon presentation, the dog exhibited lethargy, severe respiratory distress, and diffuse subcutaneous emphysema. Physical examination revealed pale mucous membranes, tachycardia (180 bpm), hypothermia (33.5 °C), and dry, crackling pulmonary sounds localized to the left thorax. Paradoxical movement of the ribcage on the left side during inspiration further indicated the presence of flail chest. Thoracic radiographs revealed pneumothorax, pneumomediastinum, atelectasis of the caudal left lung lobe, and fractures of the 4th and 5th ribs (Figure 1). Due to the unavailability of on-site CT imaging, further advanced diagnostic evaluation of the thoracic injuries was not possible. Complete blood count and serum biochemical profile revealed HCT within the normal ranges (48%; reference range: 37.3 – 61.7), leukopenia (3.28 x 10^3/μL; reference range: 6-15 x 10^3/μL), thrombocytopenia (110 x 10^3/μL; reference range: 180-484 x 10^3/μL), hypoproteinemia (3.9 g/dL; reference range: 5.2-8.2 g/dL), hypocalcemia (6.8 mg/dL; reference range: 8.5-11.9 mg/dL), and elevated alanine aminotransferase (ALT) (245 IU/L; reference range: 10-94 IU/L).

The patient was admitted to the Intensive Care Unit (ICU), and stabilization measures were initiated. To alleviate respiratory distress, thoracentesis was performed, removing 500 mL of air. Treatment included flow-by oxygen, intravenous crystalloids (Lactated Ringer's), a constant rate infusion (CRI) of fentanyl (0.1 μg/kg/min; Fentanyl®, Demo S.A.) and lidocaine (20 μg/kg/min; Xylozan®, Demo S.A.), along with intramuscular (IM) administration of pethidine (3 mg/kg, TID; Petidina Cloridrato®, Molteni) and paracetamol (10 mg/kg, BID; Apotel Plus®, UNI-PHARMA). Additionally, a fentanyl transdermal patch (50 μg/h, Fentadur®, Lavipharm) was placed. Samples for bacterial culture were obtained from the wounds located on the left thorax.

Figure 1. Pneumothorax, pneumomediastinum, atelectasis of the caudal left lung lobe and subcutaneous emphysema.

Antibiotic therapy was initiated, pending culture results, with enrofloxacin (5 mg/kg, SID; Baytril®, Bayer), clindamycin (10 mg/kg, SID; Dalacin C®, Pfizer), and cefuroxime (20 mg/kg, BID; Zinacef®, Sandoz) administered intravenously (IV). A urinary catheter was also placed to monitor urine output.

Initial clinical improvement was noted, with stabilization of the heart rate, respiratory rate, and temperature. However, the following day, the subcutaneous emphysema was exacerbated, the dog developed severe dyspnea, and repeated thoracenteses were required, removing a total of 2 liters of air.

Due to persistent and substantial air accumulation, thoracentesis alone was deemed insufficient, and a single ABPP was performed using 5 mL/kg whole blood as described in veterinary literature (Oppenheimer et al., 2014). This involved the collection of 35 mL of the patient's blood via the jugular vein in syringes, followed by immediate instillation into the left pleural cavity under sterile conditions. Initially, the procedure was well tolerated with no immediate complications and improvement of dyspnea. Before the procedure, a CBC was repeated, revealing a downward trend of HCT (37%).

Approximately 12 hours post-ABPP, the dog exhibited worsening respiratory distress and tachypnea, accompanied by ECG abnormalities ranging from multiple ventricular premature complexes (VPCs) to ventricular tachycardia (VT). Newly obtained radiographs revealed the presence of a severe pneumothorax (Figure 2). A TT size 18 Fr (Thoracic drainage catheter without trocar, Polymed, India), was placed on the left thorax, enabling continuous suction for 24 hours to further manage the pneumothorax. For TT placement fentanyl (3 μg/kg IV) was administered for premedication, and propofol (at a total dose of 3 mg/kg IV) for induction. Intercostal block was performed with lidocaine (2 mg/kg). An increased dose of lidocaine CRI (80 μg/kg/min/) was initiated for analgesia and management of VT. Furthermore, intrathoracic administration of (1 mg/kg, QID; Ropivacaine Hydrochloride, B. Braun) via the TT was added to the analgesic plan.

Forty-eight hours post TT placement, only 30 mL of air was aspirated, and the TT was kept in place for an additional 24 hours. During this time, the dog was eupneic with normal mental and cardiovascular status and a gradual return to oral intake of food and water.

Seven days after ICU admission, radiographic re-

Figure 2. Pneumothorax and increased radiopacity in the thorax due to the blood patch.

Figure 3. Resolved pneumothorax two days before discharge.

evaluation showed no further air accumulation (Figure 3). New bloodwork revealed anemia (HCT 20%), thrombocytopenia (92 x 10^3/μL), and mild elevation of liver enzymes (ALT 160 IU/L; reference range: 10-94 IU/L and ALP 126 IU/L; reference range: < 90 IU/L). Previously noted biochemical abnormalities were within normal limits. Two days later, the dog was discharged from the clinic with analgesic and antibiotic therapy, along with instructions for movement restriction.

Regular updates from the owners and the referring veterinarian confirmed the dog's good clinical condition post-discharge. At the time of the last follow-up (2 years after treatment), he remains asymptomatic and free of complications.

CASE 2 (C2)

A 10-month-old intact, 2.7 kg, female domestic short-haired cat was presented to our clinic for evaluation and management of chronic respiratory distress. History revealed trauma sustained seven months before presentation. Clinical examination findings included normal mental status, pale mucous membranes, tachycardia (190 bpm), tachypnea, abdominal breathing, paradoxical movement, and dysmorphia of the thoracic wall, especially on the right side.

Radiographs revealed loss of diaphragmatic continuity, increased intrathoracic density, and pleural effusion, supporting a diagnosis of diaphragmatic hernia (Figure 4). Biochemical profile was unremarkable, and CBC revealed a HCT of 32% (reference range: 30-44). The hernia was surgically repaired on

Figure 4. Loss of diaphragmatic continuity, increased intrathoracic density, and pleural effusion.

the same day through a midline laparotomy, revealing that the left liver lobes, the jejunum, and part of the omentum had protruded through a defect on the left side of the diaphragm into the thoracic cavity. During the procedure, no adhesions were observed between the herniated organs and the intrathoracic structures. At the end of the surgery, thoracentesis was performed to remove the accumulated air.

Following the procedure, the cat was admitted to the ICU, and a therapeutic protocol was initiated, consisting of flow-by oxygen administration, intravenous fluid therapy with Lactated Ringer's solution, furosemide (2 mg/kg, BID, IV; Lasix®, Sanofi-Aventis), pethidine (2 mg/kg, BID, IM) and meloxicam (0.05 mg/kg, SID, SC; Metacam®, Boehringer Ingelheim). Initial improvements in heart and respiratory rate were observed.

However, 24 hours after surgery, the cat developed severe dyspnea and tachypnea. Thoracic radiographs revealed atelectasis of the caudal right lung lobe and pneumothorax (Figure 5). An initial thoracentesis removed 170 mL of air, alleviating the respiratory symptoms temporarily. Despite repeated thoracenteses, continuous and substantial air accumulation necessitated the placement of a small bore tube size 6 Fr (Guidewire Chest Drain; MILA International, USA), using the modified Seldinger technique. For the tube placement topical anesthesia with local infusion of lidocaine (2 mg/kg) was performed and fentanyl was administered as a bolus (2 μg/kg, IV). Additionally, a single ABPP with 15 mL (5 mL/kg) of autologous blood was performed through the TT to enhance pleural defect closure, as previously mentioned in the literature (Oppenheimer et al., 2014; Théron et al., 2021).

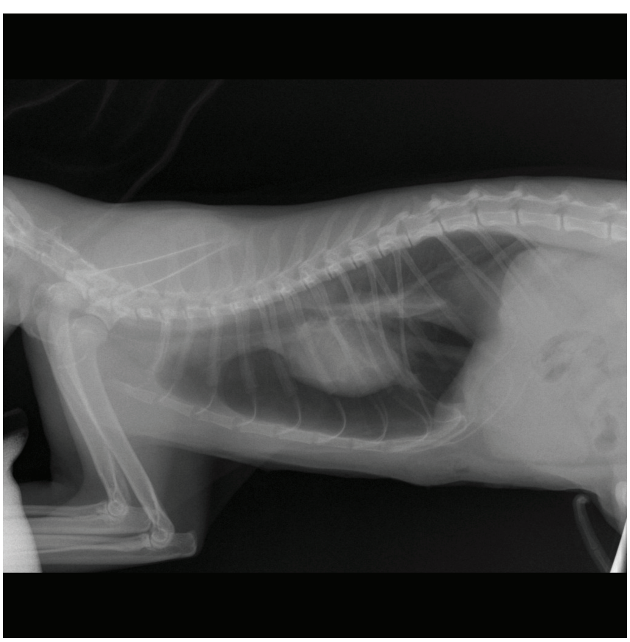

Figure 5. Atelectasis of the caudal right lung lobe and severe pneumothorax.

The cat's poor cardiopulmonary condition and persistent air accumulation in the thoracic cavity raised suspicion of a ruptured pulmonary bulla. Due to the unavailability of on-site CT imaging and the high risk associated with patient transfer due to her critical condition, a definitive diagnosis could not be confirmed. A new CBC revealed an HCT of 26%, leading to the decision against a second ABPP. Consequently, an exploratory thoracotomy was performed via the right 6th intercostal space to address any pulmonary defects. Identification of the air leakage proved challenging, particularly in the absence of CT guidance. The thoracic cavity was filled with warmed sterile saline solution and the lungs were inflated in order to detect the area of air leak. After a prolonged search, it was located on the rostromedial mediastinal surface of the right caudal lung lobe, which was subsequently resected. During this prolonged procedure, low oxygen saturation and severe hypercapnia were noticed (end-tidal carbon dioxide up to 110 mmHg), which failed to respond to intermittent positive pressure ventilation. Post-operatively, although pneumothorax was successfully managed, the cat's clinical condition deteriorated significantly, presenting with a comatose mental status. Brain damage due to prolonged hypoxia during thoracotomy was deemed the most likely cause.

Therapeutic measures were directed to reduce intracranial pressure and included flow-by oxygen, intravenous fluids (0.9% NaCl along with 3.75% hypertonic NaCl), 20% mannitol (0.5 g/kg IV), and dexamethasone (0.2 mg/kg, IV; Dexamethasone®, Fresenius Kabi). The cat remained comatose for an additional 24 hours and ultimately succumbed.

DISCUSSION

In the present study, two cases of pneumothorax of different etiology are described. Traumatic pneumothorax, as described in the dog of case 1, is considered the most common type of pneumothorax in veterinary patients (Pawloski & Broaddus, 2010). On the other hand, C2 deals with a cat that developed pneumothorax as a post-surgical complication of a diaphragmatic hernia repair. Pneumothorax is a commonly reported complication of chronic diaphragmatic hernia surgical repair, especially in the presence of adhesions (Minihan et al., 2004). Lung laceration following division of the adhesions is not unusual in such cases requiring a partial lung lobectomy. However, in this case, no adhesions were evident during herniorrhaphy. Furthermore, the delayed onset of symptoms and air accumulation (24 hours post-operatively) suggests a dynamic process rather than an immediate surgical complication. The air leak identified on the right caudal lung lobe was anatomically distant from the left-sided diaphragmatic defect, further excluding a direct connection of the hernia repair with the intrathoracic air accumulation. Bullae or pulmonary pseudocysts have been associated with trauma in small animals (Bertolini et al., 2020; Mulholland & Keir, 2019). Based on these findings and considering the cat's trauma history, we hypothesize the rupture of a pre-existing pulmonary bulla due to post-operative changes in intrathoracic pressure and lung re-expansion as the most probable cause of pneumothorax in this cat.

Both cases were initially managed with thoracentesis. In the case of C1, due to the dog's poor clinical condition and the need for repeated thoracenteses within 24 hours of diagnosis, an ABPP was performed. The decision to prioritize ABPP over TT placement was based on the fact that the dog had a flail chest, and TT placement was technically challenging due to marked regional deformation caused by bite wounds and the risk of further injury to the pulmonary parenchyma. Initial improvement was succeeded by a rapid decline in cardiopulmonary status, so TT placement and continuous suction were considered inevitable, leading to significant clinical improvement until discharge. In C2, a TT was placed, followed by ABPP to address a persistent air leak post-surgery. Due to the rapid intrathoracic air accumulation, deterioration of the animal's condition, and unavailability of CT imaging on site, exploratory thoracotomy was ultimately required to identify the source of the air leakage. According to human thoracic surgery guidelines, surgical intervention is recommended as a treatment option for patients with secondary spontaneous pneumothorax if an air leak persists for over 48 hours (MacDuff et al., 2010). Thoracotomy and lung lobectomy have been reported to achieve an 88% success rate in the treatment of persistent pneumothorax in dogs (Puerto et al., 2002).

ABPP is a more recently described technique in veterinary medicine for the management of pneumothorax (Oppenheimer et al., 2014) and other pleural disorders (Lombardo & Weatherton, 2021). It has been hypothesized to promote pneumothorax resolution through a twofold process: it induces pleural inflammation that encourages adhesion between the parietal and visceral pleura, while also forming a coagulated blood patch that seals the air leak (Merbl et al., 2010; Théron et al., 2021). Other agents, such as talc, tetracyclines, and silver nitrate, have been used to induce pleurodesis in veterinary medicine, but these methods are associated with higher complication rates and generally unsatisfactory outcomes (Jerram et al., 1999; Mitchem et al., 1999). Although the ideal volume of blood for ABPP has not been definitively established, retrospective evaluations in dogs and cats recommend a dosage of 5-10 mL/kg (Oppenheimer et al., 2014; Théron et al., 2021). Following these guidelines, approximately 5 mL/kg was used in the present clinical cases, applied to one hemithorax per animal.

While studies on ABPP in veterinary medicine are promising, the number of published reports remains limited. A retrospective study involving 8 dogs with traumatic and spontaneous pneumothorax reported an 87.5% success rate (Oppenheimer et al., 2014). Another study examining 5 dogs with persistent pneumothorax of various etiologies showed an 80% success rate following a single ABPP procedure (Théron et al., 2021). Cornel *et al.* (2020) reported 78,2% rate of pneumothorax resolution in 28 cases that increased to 100% following a second patch procedure. Notably, the same case series included a successful application of ABPP in a horse (Cornel et al., 2020). Several other successful individual cases in dogs have also been documented for cases of pneumothorax secondary to metastatic osteosarcoma (Moses et al., 2023), pulmonary mast cell tumor (Shinsako et al., 2022), and diaphragmatic herniorrhaphy (Merbl et al., 2010). The use of ABPP has also been documented in cats (Satmary et al., 2023; Wong & Mooney, 2020). The ABPP also has the potential to be used in various other conditions. It has recently been described as a successful treatment option for persistent urethral leakage in a dog (McCord et al., 2024).

The application of ABPP is associated with a low complication rate in both human and veterinary medicine. Empyema is the most commonly reported complication in human medicine, followed by fever and pleural effusion (U. Cagirici, 1998). Similar adverse effects have been observed in canine patients (Oppenheimer et al., 2014). While antimicrobial therapy is often required in animals with pneumothorax due to underlying conditions, its routine use following blood patch application is not currently justified based on available evidence. In our study, antibiotic therapy was administered to the dog (C1), as an infection was confirmed by a positive culture due to bite injuries.

In C1, the dog experienced a decrease in HCT, from 37% prior to the blood patch to 20% after the blood patch. While the blood collection used for the patch likely contributed to this side effect, it is unlikely to be the sole cause because this dog was a trauma patient with other concurrent possible sites of blood loss (skin wounds, pulmonary contusions, increased vascular permeability associated with shock etc.). Similarly, in C2, the cat exhibited a reduction from 32% (before herniorrhaphy and ABPP) to 26% (after ABPP). This reduction in HCT led to the decision against using higher blood volumes or a second patch session, which may have provided more immediate and favorable results in both cases. Veterinary literature includes examples where higher volumes (up to 10 mL/kg) or multiple patch sessions (even up to three) were necessary for the resolution of the pneumothorax (Oppenheimer et al., 2014; Théron et al., 2021). In cases where the clinicians believe that an ABPP will exacerbate the patient's condition, allogenic (Bersenas & Hoddinott, 2020; Moloney et al., 2022) or even xeno-blood patch pleurodesis (Thyen et al., 2024) have demonstrated promising outcomes and could be considered as an alternative.

Twelve hours following ABPP, the dog's (C1) respiratory and cardiovascular status deteriorated significantly. We hypothesize that the instillation of blood into the pleural cavity effectively sealed defects in the parietal pleura responsible for air leakage, such as subcutaneous emphysema or air escape, but likely failed to seal defects in the visceral pleura, leading to tension pneumothorax. The optimal timing for ABPP initiation remains unclear and is determined on a case-by-case basis. Evman et al. (2016) proposed performing ABPP in patients with refractory pneumothorax 48 hours following thoracostomy to limit additional expenses. Théron et al. (2021) suggest, for ABPP, a maximum delay of three days to allow conservative therapy to take effect before surgical intervention. The successful use of ABPP as early as 13 hours after pneumothorax diagnosis has been previously documented in a feline patient (Wong & Mooney, 2020). In C1, approximately 50 hours post-ABPP, pneumothorax resolution was achieved. While the exact contribution of ABPP remains uncertain, the patient fully recovered and remains asymptomatic. We propose that in cases where ABPP is conducted without a TT in place, close monitoring is essential. A significant limitation in both of our cases, particularly in C2, was the lack of CT imaging to identify the source of the air leak. This information could have significantly shortened the duration of exploratory thoracotomy and potentially contributed to more favorable outcomes. Furthermore, the location of the air leak in C2 (rostromedial mediastinal side of the right caudal lobe) could explain the patch failure, as the blood may not have reached the site to achieve a seal, particularly given the small volume of blood used.

In conclusion, ABPP, particularly when combined with thoracostomy tube placement and continuous suction, can be an effective and minimally invasive technique for managing pneumothorax. It is a viable option in cases of persistent air leaks where patients are poor candidates for anesthesia or surgery (e.g., due to diffuse pulmonary disease) or when financial constraints limit prolonged hospitalization. Despite these advantages, ABPP should not be regarded as a substitute for surgical intervention in cases where surgery is indicated. Additionally, the use of computed tomography can play a significant role in diagnosis and clinical decision-making, potentially improving patient outcomes.

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