

## One more 'T' for the mnemonic of the management of pulseless electrical activity: tension pneumoperitoneum

處理無脈搏電流活動助記法再多一個「T」：張力性氣腹

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The approach in Advanced Cardiac Life Support provided by the American Heart Association and the European Resuscitation Council guidelines for the management of pulseless electrical activity emphasizes on the search of reversible causes. Tension pneumoperitoneum is an uncommon surgical emergency encountered in the accident and emergency department. We report a case of pulseless electrical activity secondary to tension pneumoperitoneum in an elderly woman. Restoration of spontaneous circulation was immediately achieved after needle decompression. The management of tension pneumoperitoneum is discussed. (*Hong Kong j.emerg.med.* 2009;16:237-241)

美國心臟協會及歐洲復甦議會提供的高級心臟生命支持方法，對處理無脈搏電流活動的指引，強調尋找可逆轉的成因。張力性氣腹是急症室不常見的外科急症。我們報告一名老婦因張力性氣腹引致無脈搏電流活動的個案，針刺減壓後立即成功恢復自發性的循環。本文亦討論張力性氣腹的處理。

**Keywords:** Abdomen, compartment syndromes, heart arrest, pneumoperitoneum

**關鍵詞：**腹部、腔隙綜合徵、心臟停頓、氣腹

### Introduction

Pulseless electrical activity (PEA) is defined as the presence of organised electrical activity of the heart but no pulse. According to the current Advanced Cardiac Life Support (ACLS) guidelines of the American Heart Association (AHA)<sup>1</sup> and the European Resuscitation Council guidelines,<sup>2</sup> the management of PEA stresses on performing high-quality cardiopulmonary resuscitation (CPR) with minimal

interruptions and identifying reversible causes or complicating factors. The AHA summarises those potentially reversible conditions as "H"s and "T"s ("H"s stand for hypovolemia, hypoxia, hydrogen ion, hyper/hypokalemia, hypothermia and hypoglycemia. "T"s stand for toxins, tamponade, tension pneumothorax, coronary thrombosis, pulmonary thrombosis and trauma).<sup>1</sup> Acute abdominal compartment syndrome secondary to tension pneumoperitoneum can cause rapid cardiorespiratory decompensation.<sup>3</sup> It is due to the occlusion of the venous return from the inferior vena cava by the excessive intraperitoneal pressure and the difficulty in ventilation by the cephalic displacement of the diaphragm.<sup>4,5</sup> Early recognition and prompt decompression of the intraabdominal pressure (IAP) can result in immediate and dramatic resolution of the cardiovascular collapse. We report a case of sudden cardiac arrest with PEA secondary to tension pneumoperitoneum which was not proffered by the

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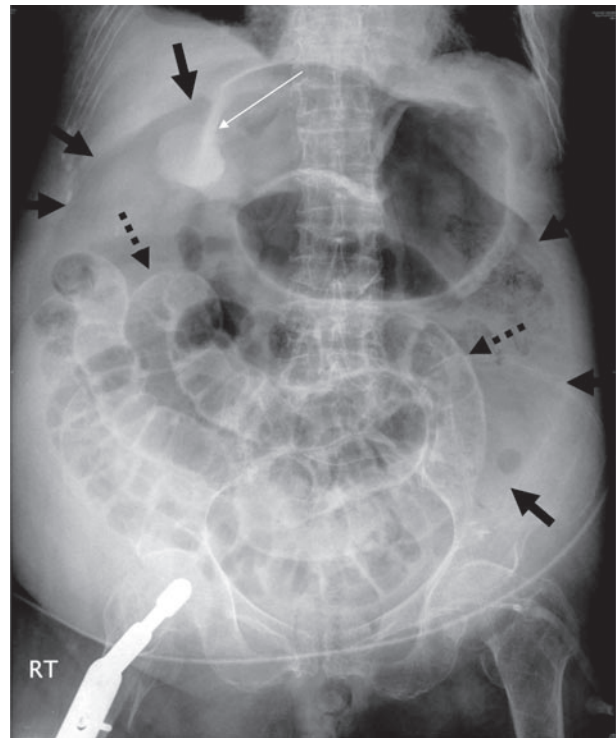
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current resuscitation guidelines as "one of the potentially reversible causes" for PEA. Literatures on the topic are also reviewed.

## Case

A 93-year-old lady with known history of hypertension and dementia was brought to the accident and emergency department in December 2008 because of increasing abdominal distension and shortness of breath for one day. She was discharged half month ago after an uncomplicated operation for fracture of her right femur. On arrival, she was found to have grossly distended abdomen with sluggish bowel sounds. Her initial blood pressure was 158/89 mmHg with a pulse rate of 90 bpm. She was in respiratory distress with a respiratory rate of 30 breaths per minute and the SpO<sub>2</sub> was 99% on oxygen supplement via facial mask. The spot bedside glucose level was 7.7 mmol/L. The electrocardiogram (ECG) showed atrial fibrillation. The patient was sent for urgent chest X-ray and abdominal X-ray imaging. However, she developed cardiac arrest shortly after the imaging. She was immediately sent to the resuscitation room. The initial ECG showed asystole. Cardiopulmonary resuscitation was performed according to the standard ACLS algorithm. She was intubated and boluses of intravenous adrenaline were administered. About 5 minutes after the resuscitation, she started to have PEA. Possible reversible causes for PEA were sought. The arterial blood gas analysis showed metabolic acidosis with a pH of 6.97. The blood potassium level was normal. Clinically, there was no evidence of cardiac tamponade or tension pneumothorax. Sodium bicarbonate solution was infused in view of the severe metabolic acidosis. The patient had persistent PEA despite continuation of CPR for 10 minutes. At this point, the abdominal X-ray was available and revealed radiological signs of pneumoperitoneum including the football sign,<sup>6</sup> Rigler's sign,<sup>7</sup> and visualisation of the extrahepatic segment of the ligamentum teres<sup>8</sup> (Figure 1). Tension pneumoperitoneum with abdominal compartment syndrome was suspected based on the grossly distended abdomen with radiological evidence



**Figure 1.** Supine abdominal X-ray showing signs of pneumoperitoneum including Rigler's or "double wall" sign (dotted black arrows); "football or dome sign" (outlined by thick black arrows); and ligamentum teres sign (thin white arrow).

of pneumoperitoneum, poor lower limb circulation with cyanosis and ineffective ventilation via the endotracheal tube. At around 20 minutes post-cardiac arrest, percutaneous needle decompression of the abdomen was performed with a gush of air returned and straw coloured fluid drained out. A nasogastric tube was inserted to decompress the stomach. Restoration of spontaneous circulation (ROSC) was immediately achieved with the blood pressure at around 120/70 mmHg. She was arranged to be admitted for further management after urgent surgical consultation. Around 30 minutes after the first attempt of needle decompression, the patient's vital signs deteriorated with decreasing blood pressure and she subsequently developed PEA again. On examination, the abdomen was tense. Needle decompression was performed again with prompt achievement of ROSC. The blood pressure was maintained at around

100/70 mmHg. She was transferred to the surgical ward for further management. The case surgeon discussed the option of laparotomy with the patient's relatives. High risk of operation and poor prognosis were explained to them. The relatives opted for no surgical intervention and no further resuscitation. The patient was managed conservatively with mechanical ventilation and haemodynamically supported with inotrope. She finally succumbed around one hour after admission to the surgical unit.

## Discussion

Aetiologies of tension pneumoperitoneum reported in the literature can be divided into gastrointestinal (GI), pulmonary, and combination of GI and pulmonary causes. GI causes result from pathological or iatrogenic rupture of the GI tract, like post-endoscopy,<sup>9-16</sup> perforated peptic ulcer (PPU)<sup>17-19</sup> and aerophagia<sup>20</sup> etc. Cases of pneumoperitoneum secondary to gastric rupture during cardiopulmonary resuscitation have been reported.<sup>21,22</sup> Mechanical ventilation was the main culprit of the pulmonary causes of tension pneumoperitoneum.<sup>23,24</sup> Positive pressure ventilation may induce barotrauma to airways and alveoli, causing air leak to perivascular spaces towards the mediastinum. Air may subsequently travel through areas of weakness of the diaphragm, i.e. the foramina of Morgagni and Bochdalek, into the peritoneal cavity, causing pneumoperitoneum.<sup>25</sup>

Rapid elevation of IAP due to tension pneumoperitoneum can result in acute abdominal compartment syndrome. Increased IAP results in numerous physiological changes decreasing the cardiac output.<sup>26</sup> Elevation of both diaphragms increases intrathoracic pressure and reduces lung compliance. This manifests as pulmonary hypertension and ventilation-perfusion mismatch, leading to hypoxia, hypercapnia and acidosis. Preload is abated by several mechanisms. Abdominal pressure is transmitted to the retroperitoneal veins, causing distal pooling of blood. The elevated diaphragm also distorts the crural vena cava, leading to functional obstruction of venous blood flow. The ROSC in our case shortly after decompression could be explained by the enhanced venous return. Other possible physiological outcomes of increased IAP are summarised in Table 1.

The pneumoperitoneum in our patient could be due to perforation of the GI tract e.g. PPU. Persistent leakage of air might account for the recurrent episode of tension pneumoperitoneum. However, post-mortem examination was waived and hence no actual pathology was confirmed. Pneumoperitoneum could be detected by typical radiological findings in the supine abdominal X-ray including right upper quadrant hyperlucency over the liver, Rigler's sign (double wall sign), falciform ligament sign, telltale triangle sign and football sign etc.<sup>27</sup> The football sign as illustrated in our patient represents the presence of a significant amount of intra-abdominal free gas.

**Table 1.** Physiological outcome of increased intra-abdominal pressure<sup>26</sup>

Increased	Decreased
Pulmonary capillary wedge pressure	Cardiac output
Peak airway pressure	Venous return
Central venous pressure	Visceral blood flow
Pleural pressure	Renal blood flow
Inferior vena cava resistance	Glomerular filtration rate
Renal vein pressure	Abdominal wall compliance
Systemic vascular resistance	

Tension pneumoperitoneum should be a diagnosis made clinically. Haemodynamic instability and respiratory distress in the presence of grossly tympanic abdominal distension should raise clinical suspicion. Prompt diagnosis is the key to potentially life-saving decompression. The decompression technique used in our case was relatively simple. We inserted a 14-gauge catheter under aseptic technique to the left lower quadrant of the abdomen. A large gush of air resulted with dramatic recovery of the cardiovascular status.

Our patient finally succumbed after admission. It could be due to fatal sepsis secondary to unrepaired perforated GI tract. Raised IAP could also jeopardize the blood flow to multiple organs. It might cause multi-organ failure<sup>28</sup> and bowel ischaemia, and both would have high mortality.

Cases with good recovery from early decompression of tension pneumoperitoneum have been reported in the literature. Peppriell and Bacon reported a patient with PEA due to tension pneumoperitoneum following colonoscopy. Needle decompression was performed about five minutes after the start of resuscitation and vital signs immediately returned to pre-arrest levels. The patient was subsequently admitted to the intensive care unit and discharged on Day 10 without neurological deficit.<sup>14</sup> Ng et al reported a child with tension pneumoperitoneum after pneumoreduction for intussusception leading to severe dyspnoea and somnolence that were immediately reversed by urgent needle decompression. The child was subsequently sent for laparotomy and later discharged without permanent sequelae.<sup>29</sup>

## Conclusion

Tension pneumoperitoneum can lead to acute cardiopulmonary decompensation and even cardiac arrest. Emergency physicians should have a high index of suspicion for this uncommon surgical emergency and keep one more "T" (tension pneumoperitoneum) in mind as one of the reversible conditions for PEA during CPR. Correct identification of the underlying pathology can result in rewarding resuscitation outcome.

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