Electrocardiographic Changes as the First Indicator of a Right Pneumothorax in an Anesthetized Child

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PNEUMOTHORAX that occurs in a patient undergoing general anesthesia may present as one of a broad spectrum of changes ranging from minimal, nonspecific hemodynamic alterations through respiratory or circulatory arrest.¹ Electrocardiographic changes as an early indicator of a pneumothorax are not widely recognized.

Case Report

A 6-yr-old, 25-kg boy with microtia of the ear was scheduled to undergo a first-stage ear reconstruction with costochondral graft placement. He had asthma since birth, and became short of breath in dusty environments. Treatment included an albuterol metered dose inhaler, the use of which was repeated twice in the last 6 months. He underwent right-sided myringotomy tube placement and general anesthesia 4 months earlier without incident. Physical examination was unremarkable except for microtia. Blood pressure was 96/42 mmHg, heart rate was 115 beats/min, and hematocrit was 56%.

After premedication with 2.5 mg nebulized albuterol and 10 mg oral midazolam, the patient was taken to the operating room, where a noninvasive blood pressure cuff, precordial stethoscope, pulse oximeter, and electrocardiograph leads II and V₅ were applied, as is the author’s practice. A preinduction electrocardiographic tracing was recorded (fig. 1A), and inhalational induction was performed using oxygen, nitrous oxide, and halothane. After placement of a 22-g peripheral intravenous catheter, 12.5 mg atracurium was given for neuromuscular paralysis, and a 5.5-mm endotracheal tube was inserted without difficulty. Breaths sounds were noted to be equal bilaterally, and end tidal carbon dioxide waveforms were present on the capnograph. Anesthesia was maintained with oxygen (FiO₂ 0.55), nitrous oxide, and isoflurane. Approximately 45 min after induction, spontaneous respiration returned, and positive-pressure ventilation was discontinued. After closure of the right costochondral graft site, the R wave of electrocardiograph lead V₅, that initially was positive was noted to be negative, and the QRS complex of electrocardiograph leads II and V₅, was increased in amplitude (fig. 1B). Auscultation of the chest revealed decreased breath sounds on the right side and no wheezing. Nitrous oxide was discontinued, and the patient’s oxygen saturation, heart rate, blood pressure, end tidal carbon dioxide, and respiratory rate remained stable. A chest radiograph confirmed the diagnosis of a large, right-side tension pneumothorax (fig. 2). The pneumothorax was evacuated using a red rubber catheter that was then removed. After evacuation of the pneumothorax, the electrocardiographic measurement returned to baseline. The remainder of the case was uneventful, and the trachea was extubated after completion. He was transported to the recovery room in good condition, and a repeat chest radiograph showed complete resolution of the pneumothorax. The patient’s postoperative course was unremarkable, and he was discharged home with no further complications.

Discussion

In several reports, researchers describe the electrocardiographic changes that may occur with a left-side pneumothorax in an awake patient.²⁻⁴ They include rightward shift in the mean frontal QRS axis, reduced precordial R wave amplitude, decreased QRS amplitude, precordial T wave inversion, and phasic voltage alternation associated with respiration. Other electrocardiographic changes with a left pneumothorax suggestive of an anterior myocardial infarction are also reported.²⁻⁴ Several mechanisms have been proposed to explain these changes. Rightward shift of the frontal QRS axis can result from clockwise rotation of the heart around its longitudinal axis as viewed from the apex, sudden increase in pulmonary vascular resistance causing right ventricular dilatation, or both. Decrease in size of the precordial R wave may result from the insulation and reduced conductance of the electrical impulse to the precordial leads caused by a retrosternal air mass, and posterior displacement or rotation of the heart.²⁻⁴ Diagnosis of a pneumothorax in an anesthetized patient presents a special challenge. The first signs, such
as tachycardia and hypotension, are usually nonspecific. As the pneumothorax expands, increased peak airway pressure and hypoxemia may develop, prompting the anesthesiologist to examine the chest. Physical examination may reveal decreased breath sounds over the ipsilateral side, expiratory wheezing, hyperresonance, tracheal shift, and subcutaneous emphysema.1

In two reports, researchers describe an electrocardiographic change as the first indicator of a left-side pneumothorax in an anesthetized adult.14,15 In each case, electrocardiographic changes were noted before any hemodynamic or respiratory aberrations. The changes included rightward shift of the QRS axis and decreased QRS amplitude. These changes were similar to those reported in awake patients with a left-side pneumothorax.

The electrocardiographic changes seen with a right-side pneumothorax in awake patients primarily involve rightward deviation of the frontal QRS axis that are less constant and less severe than those seen with a left-sided pneumothorax.1,2,5 In our case, obvious electrocardiographic changes occurred. In addition to an increase in the amplitude of the QRS complex in leads II and V5, the polarity of the QRS complex in lead V5 reversed from positive to negative. Monitoring of lead V5 in this child provided an early indication that a pneumothorax had occurred and led to prompt diagnosis and treatment before any hemodynamic or respiratory compromise. The change noted in lead II was subtle and might have gone unnoticed in the absence of simultaneous lead V5 monitoring.

These electrocardiographic changes likely result from clockwise rotation of the heart around its longitudinal axis as viewed from the apex, and from loss of the insulating effect of air to electrical conductance. The change in the direction of the QRS vector seen in lead V5 involves a shift of the transition point at which the QRS complex in the precordial leads is equiphase. This change usually occurs between V2 and V3. Figure 3
shows the position of the precordial leads and their relation to the underlying cardiac structures when the heart is positioned normally. Lead V₆, overlies the right ventricle and normally has a negative QRS complex, because the vector of left ventricular depolarization is traveling away from it. Leads V₅ and V₄ overlie the left ventricle, and the QRS complex is positive because the vector of left ventricular depolarization is traveling toward them. We believe that clockwise rotation of the heart placed the right ventricle under lead V₅, resulting in a negative QRS complex because the vector of left ventricular depolarization was traveling away from lead V₅.

With the heart in its normal position, air in the left lung provides an insulating effect for the conduction of electrical impulses to leads placed on the left chest. As seen on the radiograph (fig. 3), a leftward shift of the heart would place it closer to the leads placed on the left chest, eliminating the insulating effect of air and causing an increase in the amplitude of the QRS complex in leads II and V₅. During evacuation of the pneumothorax, note the alternation of the size of the QRS complex in leads II and V₅ and reversal of the QRS complex in lead V₆ (fig. 1B). This phenomenon is probably secondary to anatomic oscillations in cardiac position, as suggested originally by Kuritzky.⁷

In conclusion, electrocardiographic changes and, in particular, the change in electrocardiogram lead V₅, may allow prompt diagnosis of a pneumothorax and are potentially useful in children at high risk for this complication.

References

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